वक्रतुण्ड महाकाय
सूर्यकोटि समप्रभः।
निर्विध्नम कुरुमे देव
सर्व कार्येषु सर्वदा ।।

सर्वमंगलमांगल्ये
शिवेसर्वार्थसाधिके।
शरणये त्रयंबकेगौरी
नारायणी नमोस्तुते।।

THIS PIECE OF WORK IS DEDICATED
TO
THE INNOCENT TINY
SUBJECTS
OF
THIS STUDY

ROLE OF CRANIAL SONOGRAPHY IN NEONATES AND INFANTS

THESIS
FOR
DOCTOR OF MEDICINE
(RADIODIAGNOSIS)

BUNDELKHAND UNIVERSITY
JHANSI (UP)

2003 **MOHIT AGARWAL**

CERTIFICATE

This is to certify that the work entitled "ROLE OF CRANIAL SONOGRAPHY IN NEONATES AND INFANTS" which is being submitted as a thesis for M.D. (Radiodiagnosis) Examination 2003, of Bundelkhand University, has been carried out by **Dr. Mohit Agarwal** under my direct supervision. The techniques embodied in this thesis were undertaken by the candidate himself and the observations recoded were checked and verified by me from time to time.

Dated: 28.4.03

Prof. A.K.Gupta MD, MICR
Head of the Department,
Department of Radiodiagnosis,
MLB Medical College,
Jhansi

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Dated: 30/4/03

Dr. Ganesh Kumar MD
Associate Professor,
Department of Radiodiagnosis,
MLB Medical College,
Jhansi
(Guide)

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Dated: 20.4.03

Dr. Lalit Kumar MD, DCH
Lecturer,
Department of Pediatrics,
MLB Medical College,
Jhansi
(Co-Guide)

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Mohit

Dated: 30.04.2003

Mohit Agarwal

CONTENTS

INTRODUCTION

INTRODUCTION

The most crucial phase for a child, one that has the deepest impact on his life expectancy is the neonatal period to first year of life. Infant mortality rate is thus, aptly, the most important indicator of the health status of a community. The most important component of infant mortality and the one that constitutes its major part is the neonatal mortality. In India, the commonest causes of neonatal mortality are prematurity and low birth weight.

Cerebral intraventricular hemorrhage is one of the principal causes of death among prematurely born infants, being found at autopsy in 1/3 to ½ or more of infants weighing less than 2,000 gms at birth [1]. Papile et al [2] reported the incidence of intraventricular hemorrhage to be 43% in infants <1500gms in 1978. More recent reports [3, 4, 5] have shown a declining but still substantial incidence of about 20-25%. The incidence of periventricular leukomalacia has been reported to range from 2.3% to 26% [6, 7] in preterm infants and has been demonstrated in up to 75% of postmortem examinations. Other detected anomalies include ventriculomegaly, several cystic lesions, congenital abnormalities and masses.

With increasing therapeutic advancements, the diagnostician owes a great responsibility to the treating physician and to the society as a whole; to provide a prompt expert opinion.

Early and accurate diagnosis of intracranial abnormalities in neonatal period and infancy helps a great deal in proper management of high risk babies and contributes to a reduction in neonatal and infant mortality.

In the late 1970s and early 80s, an analysis of multiple clinical features in infants shown to have periventricular-intraventricular hemorrhage by CT scan showed poor sensitivity, specificity and predictive value. This finding established the need for a screening tool, which has been filled by cranial ultrasonography [8].

The role of ultrasound to detect intracranial hemorrhage in neonates first came to attention in 1979. The earliest published photographs were produced by linear array scanners and were made in the axial plane imaging through temporal and parietal bone [8]. Much artifact was apparent and resolution was poor because longer wave length, higher energy ultrasonic waves were needed to penetrate the skull.

Cooke suggested these problems can be overcome by using a sector scanner and imaging through the anterior fontanelle [8].

Current ultrasound technology allows for rapid evaluation of neonates and infants in the intensive care nursery with virtually no risk [9].

Non-invasive, rapid evaluation of brain in the neonate and infants with reproducible results is now feasible with real-time high resolution cranial sonography through the anterior fontanelle. Cerebral anatomy in coronal and sagittal planes is visualized and the entire ventricular system and adjacent brain parenchyma are identified. The procedure is rapid and can be done in the incubator making transport of sick neonates unnecessary. Ventricular size, intracranial hemorrhage, abnormalities of the ventricular system, subdural effusion and cystic lesions are recognized by the usual sonographic criteria [10].

Besides anterior fontanelle, supplemental imaging windows have developed for better assessment of brain. Posterior fontanelle imaging allows improved detection of IVH. This technique better demonstrates subtle differences in echogenecity between clot and a choroid glomus and depicts clot extending into occipital and temporal

horns. Mastoid fontanelle imaging is particularly useful in detecting hemorrhage involving the brainstem, cerebellum and subarachnoid cisterns. It greatly facilitates clot detection in the fourth ventricle and cisterna magna.

Cranial sonography is valuable in the evaluation and follow-up of periventricular leukomalacia which appears as areas of increased echogenecity in periventricular white matter, later showing cystic changes.

Sonography plays a key role in the initial evaluation and monitoring of ventricular dilatation in the newborn [11]. It reliably delineates ventricular size and anatomy in small infants. It can be useful for detecting ventriculomegaly, differentiating non-obstructive ventricular dilatation from obstructive enlargement, determining the cause of hydrocephalus, aiding in the temporary management of patients with permanent ventricular shunts [12].

In addition, solid parenchymal lesions such as diffuse hemangiomas and brain tumors are depicted as changes in sonographic parenchymal architecture [11].

Cranial ultrasonography offers an excellent anatomic imaging of the brain when evaluating for congenital anomalies and plays an important role in the diagnosis of inflammatory processes [13].

REVIEW OF LITERATURE

REVIEW OF LITERATURE

GERMINAL MATRIX HEMORRHAGE

Germinal Matrix Hemorrhage (GMH) is a common event occurring primarily in premature infants those are less than 32 weeks' gestational age.

Volpe JJ in 1989 stated that the risk factors for GMH include both perinatal and postnatal events [17]. Several authors have noted that the pathophysiology of early onset GMH in the first 6-12h of life may differ from that of later onset hemorrhage. One such study was done in 1984 by Ment et al [18]. Vaginal delivery, labor and intrapartum asphyxia may be related to the presence of early onset hemorrhage.

Ment LR with Oh Wb in 1992 showed that respiratory distress syndrome, vigorous neonatal resuscitation, hypoxemia, acidosis, pneumothoraces, bicarbonate administration and seizures are all risk factors for this insult [19].

The Germinal Matrix develops deep to the ependyma and consists of loosely organized, proliferating cells that give rise to the neurons and glia of the cerebral cortex and basal ganglia. The capillary bed of the

germinal matrix is composed of large, irregular vessels with little evidence for basement membrane proteins or glial supporting structures [14]. These vessels represent the "watershed zone" of the ventriculofugal and ventriculopetal vessels of the immature cerebrum and are not readily distinguishable as arterioles, venules or capillaries [15]. Grunnet [14] has demonstrated that the luminal areas of the germinal matrix vessels are significantly larger than those of cortical vessels of human fetuses of the same gestational age and has thus hypothesized that the greater diameter of these rudimentary vessels may permit greater pressure to be exerted on their walls, thus increasing their susceptibility to rupture.

Doppler studies of cerebral blood flow have suggested that the fragile germinal matrix vessels are unable to auto-regulate cerebral blood flow[2] and the combination of doppler determinations of cerebral blood flow and echoencephalography imaging have permitted the detection of increased cerebral blood flow with seizures and pneumothoraces prior to the onset of hemorrhage [16]. Finally, and perhaps of greater concern for the neurodevelopmental outcome, both 133 Xenon inhalation studies of cerebral flow [17] and positron emission tomography studies [21] have documented prolonged diffuse depression of cerebral blood flow throughout the cerebral

hemispheres in infants with even low grade hemorrhage. Thus, clinical studies suggest increases in cerebral blood flow play a role in the pathogenesis of GMH.

Since the germinal matrix begins to involute at 34 weeks of gestation, GMH and the accompanying intraventricular hemorrhage (IVH) are lesions of preterm infants. The incidence is inversely related to gestational age, and those patients with the lowest gestational ages are more likely to experience the highest grades and parenchymal involvement of hemorrhage [2]. Hemorrhages have been reported within the first postnatal hour and as many as 25% of all hemorrhages occur by the sixth postnatal hour. Over half of all preterm infants who will ever develop hemorrhage do so on the first postnatal day, and less than 5% experience hemorrhage after the fourth or fifth day of life [17].

As the fetus matures, the germinal matrix regresses towards the Foramen of Monro, so that, by full term, only a small amount is present in the caudothalamic groove. It is for this reason that GMH and IVH originate in the periventricular matrix zone located between the caudate nucleus and the thalamus at the level of, or slightly posterior to, the Foramen of Monro [17].

Various systems have been put forward to classify GMH. Mantovani (1980) [22] classified GMH into three grades: -

Grade I	:	Subependymal or Intraventricular hemorrhage filling less than 10% of ventricles.
Grade II	:	IVH filling from 10% to 50% of ventricles
Grade III	:	IVH filling 50% or greater of ventricles

Lazzara (1980) [23] classified it into mild, moderate and severe forms: -

Mild	:	SEH with or without ¼ of AP diameter of ventricles Blood filled
Moderate	:	¼ to ½ of AP diameter of ventricles blood filled
Severe	:	½ of AP diameter of ventricles blood filled

Levene (1988) [24] gave three US grades: -

Grade I	:	SEH with or without inferior or lateral extension of blood beyond most lateral border ventricles.
Grade II	:	Downward extension into basal nuclei on at least one side or involvement of caudate to genu of ventricle posterior on parasagittal scan.
Grade III	:	Large hemorrhage with any degree of extension laterally or superiorly into cerebral parenchyma.

Shankaran [25] sonographic classification for GMH is: -

Mild	:	SEH with or without small amount of blood in the ventricles (normal sized)
Moderate	:	Considerable amount of blood in enlarged ventricles.
Severe	:	Filling ventricles forming a cast or intracerebral extension of hemorrhage.

Volpe (1995) [26] revised the sonographic grading system: -

Grade I	:	SEH with/without blood in less than 10% of ventricle space
Grade II	:	SEH with blood in 10-50% of ventricular area.
Grade III	:	>50% hemorrhage filling may be accompanied With ventricular dilatation.

There was no universally accepted system, until Papile et al [27] proposed the following system of grading: -

Grade I	:	Subependymal Hemorrhage
Grade II	:	Intraventricular Hemorrhage without ventricular dilatation
Grade III	:	Intraventricular Hemorrhage with ventricular dilatation
Grade IV	:	Intraparenchymal hemorrhage with or without ventricular dilatation

Vohr et al in a review article on intraventricular hemorrhage cited the incidence of intracerebral hemorrhage to be inversely related to gestational age and those patients with the lowest gestational ages are more likely to experience the highest grades and parenchymal involvement of hemorrhage. They have also stated that hemorrhages have been reported within the first postnatal hour and as many as 25% of all hemorrhages occur by the sixth postnatal hour. Over half of all preterm infants who will ever develop IVH do so on the first postnatal day and less than 5% experience hemorrhage after the fourth or fifth day of life [16].

Clinical symptoms and signs may occur as a result of blood volume loss or neurologic dysfunction and depend, in part, on how rapidly blood loss evolves. The presentation depends on the size, site and rapidity of the hemorrhage. IVH can present as a catastrophic event when blood loss is large and rapid. Presentation can be stuttering, with intermittent periods of stabilization when there is a slower evolution of blood loss. A clinically silent presentation may occur in up to 50% of cases usually with smaller hemorrhages.

Leech et al (1974) and Volpe JJ in separate studies showed that the neuropathologic consequences of GMH include germinal matrix

destruction, periventricular hemorrhagic infarction and posthemorrhagic hydrocephalus [1, 28]. In addition, periventricular leukomalacia (PVL) is a frequent neuropathologic accompaniment of IVH but is not apparently caused by the hemorrhage itself, as stated by Ruston et al [29]. Germinal matrix destruction with secondary cyst formation is a common and expected feature of germinal matrix hemorrhages [1, 28].

Multiple studies have shown that IVH is a risk factor for subsequent motor and cognitive handicaps. Papile et al [30] reported the two year follow up of 198 premature infants with a birth weight of <1501gms. Infants with grade III and IV hemorrhage had major handicap rates of 36% and 76% respectively, whereas infants with grade I and II had major handicap rates of 9% and 11% respectively which did not differ from controls.

Subsequent investigations identified that persistent ventriculomegaly and post hemorrhage hydrocephalus were complications which contributed further to the severity of the sequelae. Krishnamoorthy et al [31] reported a relationship between ventriculomegaly at term and increased motor abnormalities at 18 months in a cohort of 228 low birth weight infants (< 1750g).

Allan et al (1982) observed that dilated ventricles occur most frequently in infants with moderate hemorrhage and may be related to post hemorrhagic obstruction, although ventricular dilatation has been noted in association with small ependymal hemorrhages [32].

More recent studies have critically evaluated the longitudinal neurodevelopmental status of low birth infants to assess effects of both low grade I and II and high grade III and IV GMH. Vohr et al (1989) prospectively evaluated a sample of 90 preterm infants with a birth weight <1750g and gestational age <34weeks and 22 normal full term controls. At 24 months of age, 75% of preterm infants with grade III-IV GMH, 93% with grade I and II GMH and 100% with no GMH were normal neurologically. These data suggest neurologic plasticity with significant motor recovery occurring in the first 2 years of life especially in infants with severe (grade III-IV) GMH [33].

Vohr with Garcia-Coll et al in 1992 [34] performed evaluation of children, diagnosed for SEH-IVH in the neonatal period, at 5 years of age, to assess cognitive function and neurologic status in low birth weight survivors. The neurologic assessment at 5 yrs revealed that 60% of children with grade III-IV GMH, 84% with grade I-II GMH, 76% with no GMH and 93% of term infants were normal.

Ischemic brain lesions may occur independent of GMH or in association with GMH. Areas of increased echogenecity consistent with ischemia may progress to cystic change or periventricular leukomalacia (PVL).

Bejar R et al (1988) observed that PVL appears to occur in the sickest infants, those with birth weights <1000g and in association with intrauterine infections [35].

Sinha et al [36] reported that 39 of 232 (17%) infants <32weeks gestation had ischemic lesions identified by cranial ultrasound. Nine infants had early onset ischemia identified within 2 hours of birth and the remaining 30 had late onset evidence of ischemia at 4-70 postnatal days. These infants demonstrated a high incidence of multiple morbidities with 67% of the early onset group and 50% of the late onset group having significant disabilities. These data suggest that infants with a combination of GMH and PVL are at greatest risk for the subsequent manifestation of neurodevelopmental sequelae.

In summary, although infants with all grades of GMH may manifest suspect or abnormal neurologic findings on examination in the first

12 months of life, data support the potential for recovery. Important predictors of recovery and a more optimal neurodevelopmental outcome include less severe grade of bleed, absence of ventricular dilatation and ischemia, shorter neonatal hospitalization and higher social and environmental status.

Because of increased risk of neurodevelopmental sequelae, multiple clinical trials have tested the effects of management techniques and pharmacologic interventions to decrease the incidence of GMH.

Studies seeking to limit blood pressure changes and therefore intracerebral blood flow fluctuations have shown beneficial effects of Phenobarbital given both postnatally and prenatally. Donn et al proved the role of phenobarbital in preterm infants in 1981 [37].

A number of recent investigators have indicated that Vitamin E administered intramuscularly or combined with oral administration may improve survival and reduce the incidence of GMH in VLBW infants. Sinha et al demonstrated a reduced incidence of periventricular hemorrhage in very preterm neonates who were given Vitamin E supplementation [38].

As a potent inhibitor of prostaglandin synthesis, Indomethacin stabilizes cerebral blood flow. In addition, it acts to promote vascularization of the germinal matrix. In 1985, Ment et al [39] reported that high dose Indomethacin in infants of 600-1250g birth weight. Side effects of decreased urinary output and fluid overload however occurred. Ment et al subsequently reported a significant reduction in GMH without side effects using low dose Indomethacin.

Ethamsylate is a water soluble non-steroidal drug which ahs been used in Europe to reduce the overall incidence of severe GMH in neonates. Morgan MEI et al showed reduced incidence of periventricular leukomalacia in very low birth weight babies treated with ehthamsylate [40].

Humble CG et al in a study on the neonatal mortality rate from respiratory distress syndrome stated that the final and perhaps most important NICU treatment of the 1990s is surfactant. The fact that Respiratory Distress Syndrome and its complications including pneumothorax have been linked to GMH and the fact that surfactant reduces the severity of RDS suggests that surfactant administration on LBW infants would be associated with a decreased incidence of GMH [41]

HYDROCEPHALUS

By definition, hydrocephalus is a dynamic abnormality in which there is active progressive increase in the volume of the ventricles, secondary to absolute, or relative, obstruction of the passage of the CSF between its origin and its absorption [42].

In humans, the cerebral hemispheres with a central lumen develop from the cephalic end of the neural tube. These represent the major brain subdivisions and the tentatively defined ventricles, both of which become further elaborated as certain regions constrict and others expand to form the basic pattern of the ventricular system.

During the second month of gestation, choroid plexi primordia develop, first as mesenchymal invagination of the roof of the fourth ventricle, then by a similar invagination of the lateral and third ventricles. By the third month, the plexi fill 75% of the lateral ventricle and then begin to decrease in relative size. As the plexi develop in the second month, the fetal ventricles are large relative to the thickness of the cortical wall, and this relative dilatation disappears with further development of white and gray matter. By the second to third trimester, the ventricles normally undergo

restricted ependymal loss without subependymal gliosis. At this time, the fourth ventricle exit foramina develop. These are the foramina of Luschka, two lateral apical roof apertures leading to pontine cistern; and the foramen of Magendie, a single median posterior roof aperture leading to cisterna magna.

The majority of CSF is produced by the choroid plexus, but it is also produced by the ventricular ependyma. CSF normally flows from the lateral ventricles, through the foramina of Monro, the third ventricle, the Aqueduct of Sylvius, the fourth ventricle, the lateral foramina of Luschka or medial foramen of Magendie, and into the basal cisterns. From there, a small quantity circulates down into the spinal subarachnoid space. CSF flows upward around the brain anteriorly and posteriorly to reach the vertex, where it is absorbed by the arachnoid granulations into the superior sagittal sinus.

The rate of CSF formation ranges from 0.3 to 0.4ml/min in children. Total CSF volume in the newborn is 50ml and increases with age to an adult volume of 150ml. At CSF pressures below 200mm H_2O, production of CSF is independent of pressure; however a prolonged and marked increase in intraventricular pressure owing to hydrocephalus can slightly reduce the rate of CSF formation [43].

Hydrocephalus is a very common entity. Milhorat (1982) observed that the incidence of approximately 3 cases per 1000 live births is an underestimation.

In most cases, hydrocephalus results from obstruction of the normal flow of CSF and thus fluid accumulates in excessive quantities within the ventricular system. Faulty absorption of CSF has also been implicated, and on occasion, so has excessive production (as in choroid plexus papilloma). These forms of ventricular enlargement must be differentiated from the ventriculomegaly seen secondary to loss of brain substance i.e. atrophy or dysgenesis [44]. If it is not possible to know whether the CSF is under pressure it is probably best to state that it is ventriculomegaly rather than hydrocephalus, so as not to imply obstruction or increased pressure [45].

In general, hydrocephalus may be classified as obstructive and non-obstructive. Obstructive hydrocephalus may be intraventricular (non-communicating type) or extraventricular (communicating type).

Intraventricular Obstructive hydrocephalus

- Obstruction at foramen of Monro
  - Post-hemorrhagic
  - Post-infectious
  - Tumor or cyst
- Obstruction at third ventricle
  - Suprasellar tumor or cyst
- Aqueductal obstruction
  - Congenital aqueductal stenosis
  - Postintraventricular hemorrhage
  - Post-infectious
  - Aneurysm of vein of Galen
  - Quadrigeminal cyst or tumor
  - Chiari II malformation (Aqueductal kink)
- Obstruction at Fourth ventricle
  - Dandy-Walker malformation (outlet obstruction)
  - Chiari II malformation (compression of fourth ventricle)

Extraventricular Obstructive Hydrocephalus

- Post-hemorrhagic
- Post-infectious
- Achondroplasia
- Absence of arachnoid granulations

Non-Obstructive

- Choroid Plexus Papilloma
- Superior vena cava obstruction
- Obstruction of Vein of Galen

Harwood-Nash DC et al state that obstruction of the aqueduct of Sylvius is the most common cause of intraventricular obstructive hydrocephalus in all age groups [42]. They observed that in the neonate and infant, most cases are developmental. Other etiologic factors include intrauterine vascular insult, infection and hypoxia.

Machado HR et al studied 100 children affected by hydrocephalus and found congenital hydrocephalus to be the most common etiologic factor. In this study Chiari II malformation was found to be the leading cause of congenital hydrocephalus.

Hayden CK [44] proposes that hydrocephalus in Chiari II malformation is due to stenosis or compression of the aqueduct of Sylvius, most frequently caused by stretching and subsequent narrowing by the downwardly displaced cerebellum.

Adams C et al [46] reported the incidence of aqueductal stenosis to be 20% of all cases of hydrocephalus. According to them the incidence ranges from 0.5 to 1.0 in 1,000 live births.

Extraventricular Obstructive Hydrocephalus most frequently results from obliteration of the subarachnoid space after hemorrhage or infection of the meninges and subarachnoid space [44].

Babcock et al proved the accuracy of ultrasound in the determination of ventricular size in 1980 [53].

Edwards and Brown et al (1981) in a prospective study of 94 real time sonographic sector scans of 56 neonates in a 6 month period diagnosed ventricular enlargement in 21 neonates. CT correlation was available in 17 cases and in no case did sonography and CT disagree as to the degree of ventriculomegaly. Generally, sonography demonstrated greater detail of the ventricular system than did CT. They noted that in the normal neonate the ventricles are frequently slit-like structures and less anatomic detail is demonstrated. The demonstration of posterior fossa structures and the brain stem is less satisfactory and the fourth ventricle is rarely seen even in the presence of ventricular dilatation [50].

Siegel et al in 1983 proposed that cranial sonography should be used as the primary examination in neonates and infants with hydrocephalus [48].

Rumack and Johnson in 1984 in a report on the types of lesion that may be better evaluated with CT scan or sonography recommended that sonography is the method of choice in hydrocephalus because it is portable, less expensive, and requires no sedation [49].

Machado HR and Machado JC et al (1985) showed that brain sonography, when used in children with patent bregmatic fontanelle, is able to; a) replace CT, except in cases suspected of harboring brain tumors; b) provide good cerebral mantle measurements and calibrate significant changes from before to after surgery, indexes which may be of value as evidence of a functioning shunt and as a prognostic factor of intellectual performance; c) be used serially as it is innocuous, inexpensive, easily performed and interpreted and accessible to the majority of neurosurgeons world wide [47].

Machado et al (1991), in a study on infantile hydrocephalus proposed that brain sonography has emerged as an effective tool in diagnosing progressive ventricle dilatation and may be used for continuous follow-up. They stated that it gives such important information as: a) cortical thickness, an expression of proper shunt function and of prognostic value concerning neuropsychological development; b) position of the tip of catheter, which is considered by some to be a

predictive factor of shunt failure; c) other complications such as subdural collections, isolated IV ventricle and slit ventricles [51].

In the evaluation of ventricle size, ratios of the width of the ventricles to standard landmarks in the skull are used.

Poland et al [52] while determining the normal values for ventricular size using real time ultrasonography in 1985, considered the lateral ventricular ratio as standard. They obtained it while scanning in the coronal plane at the level of the head of caudate nucleus and at the level of the midglomus of the choroid plexus in the ventricular atria. A ratio was obtained at both levels by taking the distance between the outer aspects of the ventricular walls and dividing it by the distance between the inner tables of the skull at this level. A ratio of more than 0.30 was considered abnormal.

Edwards et al also interpreted the scans as positive for hydrocephalus if the lateral ventricular ratio was greater than 0.30, in their study of 56 neonates [50].

Rumack CM and Manco-Johnson ML [45] consider the lateral ventricular ratio of greater than 0.34, indicative of hydrocephalus.

Johnson et al (1980) used the ventricle-to-hemisphere ratio to define hydrocephalus. The measurements are taken at the widest part of the head on the axial view; the distance from the falx to the ventricular wall is then compared to the width of the hemisphere. They reported that the ratio can be as high as 64% at 16 weeks but should decrease to less than 35% by 25 weeks [54].

Occipital horns are the first to enlarge; therefore, the measurement of atrial size is the most useful measurement of the ventricles, which is the site of confluence of the bodies, occipital horns and temporal horns. As a matter of fact the measurement of atrium actually measures the transverse diameter of the occipital horn. Cardoza reports that between 14 and 38 menstrual weeks the transverse atrial measurement is constant at 7.6 mm. Measurements above 10mm suggest ventriculomegaly [55].

Mahoney et al (1988) report that the distance between the medial atrial wall and the choroid is 1 to 2mm. Measurements of 3mm or greater were associated with abnormal outcomes [56].

Toi A and Sauerbrei EE found that the total distance between the left and right anterior ventricles never exceeds 20mm. this measurement

is less sensitive than the individual occipital measurement, but it becomes especially useful when ventricular separation is suspected as with agenesis of the corpus callosum [57].

In a study by Davies et al involving 120 infants <33weeks' gestational age had their intracranial ventricles measured. The study was done to establish normal ranges, in preterm infants <33 weeks' gestation, for measurement of the lateral, third and fourth ventricles and to assess intra-observer and inter-observer reliability. The reference ranges obtained were- anterior horn width: 0-2.9mm; thalamo-occipital distance: 8.7-24.7mm; third ventricle width: 0-2.6mm; fourth ventricle width: 3.3-7.4mm; fourth ventricle length: 2.6-6.9mm. Dependent and non-dependent lateral ventricles did not differ significantly in size. There was no clinically significant difference in ventricular size between sexes. All measurements had good intra-observer and inter-observer reliability [58].

Four major factors influence the clinical course in hydrocephalus: the time of onset, duration of increased intracranial pressure, rate at which intracranial pressure increases, and any pre-existing structural lesions. The time when hydrocephalus develops in relation to closure of the cranial sutures determines whether enlargement of the head is

the presenting sign. Before 2 years of age, progressive enlargement of the head is invariably a presenting complaint. When hydrocephalus develops after 2 years of age, any changes in head circumference are overshadowed by other neurologic manifestations. In older infants and children, the space occupying lesions responsible for hydrocephalus often produce focal neurologic signs before causing CSF obstruction [43].

The general principles of treatment are surgical correction of CSF obstruction, reduction of CSF production by drugs or surgical therapy, ventricular bypass into a normal intracranial channel in non-communicating hydrocephalus, and ventricular bypass into an extracranial compartment in either non-communicating or communicating hydrocephalus [43].

ISCHEMIC LESIONS

Ischemic cerebral injury is a frequent complication of sick newborn infants.

Weindling et al (1985) described Peri-ventricular leukomalacia (also known as White Matter Necrosis) and Focal Brain Necrosis as components of the same destructive process [59]. Porencephaly, Multicystic Encephalomalacia and Hydranencephaly comprise the continuum of Focal Brain Necrosis [59]. The idea was shared by Schuman et al [60].

Periventricular leukomalacia (PVL)

De Reuck et al (1972) proposed that PVL is a pathological term to describe the cystic changes which occur adjacent to the ventricles as a consequence of ischemia [61].

PVL was first delineated by Banker and Larroche in 1962 [69].

De Reuck et al noted that occipital radiation and the white matter around the foramen of Monro are the two most common sites for the occurrence of PVL [61].

Trounce et al (1988) and Perlman et al (1996) reported the incidence of PVL to range from 2.3% to 26% in preterm infants [6, 7].

Barson observed that PVL is found in 80% of neonatal autopsies [62]. Armstrong et al (1987) demonstrated PVL in up to 75% of post-mortem examinations [63].

Deguchi et al demonstrated that preterm infants of 22 to 30 weeks' gestation tend to experience more widespread and confluent periventricular necrosis, whereas older premature infants exhibit more focal necrosis [70].

Young et al (1982) believed the pathophysiology of PVL to involve an ischemic injury to the periventricular white matter [64].

Armstrong and Norman (1974) describe an acute phase characterized by multiple foci or coagulation necrosis in deep and periventricular white matter, and a chronic phase depicted by cavitation and scarring appearing one or more weeks after the cerebral insult [65].

Calame et al observed that in the early chronic stage multiple cavities develop in the necrotic white matter adjacent to the lateral walls of the frontal horns, body, atria, and occipital horns of the lateral ventricles [66].

Barson (1970) stated that these lesions are frequently located in the lateral wall of the atria and occipital horns causing damage to the optic radiations [62].

Sims et al (1985) noted that the cavities of PVL resolve, leaving gliotic scars and diffuse cerebral atrophy [67].

Dubowitz et al (1985) proposed that necrotic lesions with only microscopic cavities may also lead to cerebral atrophy [68].

Van de Bor et al (1992) observed that the diagnosis of cystic PVL is strongly associated with poor neurodevelopmental outcome [71].

The most frequent clinical correlate of PVL is spastic diplegia, although infants with extensive cystic PVL may show more generalized changes in tone [16].

Hirtz and Nelson observed that prenatal administration of magnesium resulted in lower incidence of PVL [72].

Porencephaly

A Porencephalic cyst is a large intraparenchymal cyst that communicates with the ventricular system [73]. Porecephalic cysts are partly, though sparsely, covered by ependyma.

Barmada et al (1979) found that porencephaly is present in term and preterm infants, but is infrequent under 30 weeks' gestational age [74].

Porencephaly results from infarction in the territory of a major artery, usually the middle cerebral artery, although at times it may be a sequel to a grade IV intraventricular hemorrhage that extends the ventricular lumen to the empty parenchymal space left by the reabsorption of the hematoma [73].

Dykes et al (1980) observed that porencephalic cysts following grade 4 IVH are visualized in survivors 10 days to 8 weeks after the event.

The clinical correlates of porencephaly are spastic hemiplegia, hemisensory deficits and often hemianopia.

Tardieu et al (1981) stated that although it is not a progressive lesion and does not obstruct the flow of CSF in chronic phase, the porencephalic cyst occasionally enlarges and causes symptoms of intracranial hypertension. [75].

In some cases, a ventriculoperitoneal shunt may be required to prevent further enlargement of the porencephalic cyst, encroachment on functional brain, and midline shift.

Multicystic Encephalomalacia

According to Menkes et al, the neonatal brain responds to infarction differently than the mature brain. Rather than forming dense gliotic scars, cysts are the usual long-term residual lesions. The reasons for the formation of cysts in the newborn brain are that areas of infarction tend to be relatively larger than in the adult because collateral circulation is less well developed and because the ability of the neonatal brain to mobilize reactive gliosis is limited [73].

Rumack et al propose that encephalomalacia is an area of focal brain damage which pathologically has astrocytic proliferation and glial septations. Typically there is no communication to the ventricular system [76].

Little et al established the relationship of cystic encephalomalacia to neonatal asphyxia [77].

Lyen et al suggested that, in some instances, fetal viral encephalitis can induce a similar pathological picture [78].

Infants surviving this type of insult usually develop a severe form of spastic quadriparesis [73].

Hydranencephaly

Dublin et al (1980) suggested that hydranencephaly could be thought of as the severest form of porencephaly [79].

In this condition, the greater portions of both cerebral hemispheres and the corpus striatum are reduced to membranous sacs composed

of glial tissue covered by intact meninges and encompassing a cavity filled with clear CSF [73].

Myers (1969) proposed that the condition is the outcome of vascular occlusion of both internal carotids, or their main branches [80]. Structures that receive a blood supply from the posterior cerebral artery or vertebral artery are spared and are usually identifiable. McElfresh et al suggested that hydranencephaly can be a consequence of intrauterine infections or other gestational insults [81].

Hockey described a proliferative vasculopathy with autosomal recessive inheritance in association with hydranencephaly [82].

Most infants with hydranencephaly do not survive beyond 23 months of life; they succumb to intercurrent infections or to an unexplained deficit of vital function.

No treatment is available for hydranencephaly.

CONGENITAL LESIONS

Babcock DS (1986) in his study on the sonography of congenital malformations of the brain described that a variety of congenital malformations can be diagnosed by sonography. He observed that the findings were very similar to those seen on computed tomography and pneumoencephalography. The study described that ultrasound images ban be obtained in multiple planes which is helpful in categorizing the lesion. Babcock concluded that since babies with congenital malformations are sometimes clinically unstable, ultrasound has the advantage of allowing diagnoses to be made without moving the infants from nursery [85].

Holoprosencephaly

Harwood-Nash et al (1976) suggested that holoprosencephaly is due to failure of the primitive prosencephalon, in part or in whole, to develop a telencephalon and a diencephalon [83].

Chawla et al (1969) proposed that in its most extreme form, holoprosencephaly manifests as Cyclops, while in milder cases only some degree of hypotelorism is noted [84].

In decreasing order of severity, holoprosencephaly may be alobar, semilobar and lobar.

Alobar holoprosencephaly demonstrates failure of visualization of the falx and a large, central, single ventricle. The thalami are fused, and a thin mantle of cerebral tissue is demonstrated, either anterior or posterior, incompletely covering the central univentricle. The third ventricle is small or absent, and the aqueduct typically small. The cerebellum and brainstem however usually are relatively normal.

With semilobar holoprosencephaly, the single ventricular cavity usually is somewhat smaller than the one seen with alobar holoprosencephaly, and there is significantly more cerebral tissue present. The third ventricle is typically incorporated into the single ventricle, although the aqueduct and fourth ventricle are normal.

Lobar holoprosencephaly manifests near normal cerebral hemispheres and thalami, but both gray and white matter remain fused with no visible falx, across the midline in the region of the frontal lobes. The frontal horns and bodies of lateral ventricles lie

close together, and this along with absence of septum pellucidum results in squared appearance of the ventricles.

Arnold Chiari Malformation

This major expression of spinal dysraphism was first observed by Cleland in 1883 but was first definitely described by Chiari in 1891.

Chiari malformations are characterized by cerebellar elongation and protrusion through the foramen magnum into the cervical spinal cord [43].

Harding B et al describe four different positions of the cerebellum and brainstem relative to foramen magnum and upper cervical canal [84].

In the Type I deformity, there is simple elongation and slight downward displacement of the cerebellar tonsils. The Type II abnormality consists of pronounced downward displacement of the cerebellum, medulla, lower pons and fourth ventricle into the spinal canal. In the rare Type III abnormality, there is abnormal

displacement of the entire cerebellum into a large cervical spina bifida and in the Type IV abnormality, there is cerebellar hypoplasia only.

Dandy -Walker Malformation

Dandy-Walker malformation described in 1914 by Dandy and Blackfan, is characterized by a triad of complete or partial agenesis of cerebellar vermis, cystic dilatation of the fourth ventricle, and an enlarged posterior fossa with upward displacement of the transverse sinuses and tentorium [43].

Barkovich et al noted that in milder cases fourth ventricular dilatation may be less striking and associated cerebellar changes less dramatic [86].

Taylor and Sanders observed that ultrasonography clearly demonstrates the large posterior fossa, small cerebellar remnant and enlarged fourth ventricle as good as CT or MRI [87].

NEOPLASMS

Harwood-Nash note that intracranial neoplasms constitute 12% to 15% of all tumors encountered in infants and children [88], and in some series they constitute the second largest group of neoplasms after leukemia.

Jooma et al observed that both supra- and infratentorial are seen in pediatric age group, but in infants, supratentorial tumors predominate [89].

Ambrosino et al proposed that in the first year of life, astrocytomas are the most common tumor type followed by choroid plexus papillomas, ependymomas, and primitive neuroectodermal tumors [90].

Han et al demonstrated that in the newborn period, teratomas as well as astrocytomas are the more common cell type [91].

The use of sonography for the detection of intracranial tumors is restricted because it relies on a patent anterior fontanelle. Consequently, it is useful only in infants and even then definition of the lesions is not as discrete as with CT or MRI.

MENINGITIS AND VENTRICULITIS

Feigen et al proposed that acute bacterial meningitis in infancy often results in significant morbidity and mortality due to cerebral involvement [92]. In a prospective study they concluded that 8% of children with bacterial meningitis suffered severe persistent intellectual or focal neurologic deficits and 28% demonstrated milder intellectual deficits.

Kirpekar et al (1986) demonstrated that CNS may be involved in very premature infants in systemic candidiasis [93].

Brown (1984) and Han BK et al (1985) demonstrated ultrasonography to be useful in the evaluation of complications of meningitis [94, 95].

The spectrum of sonographic findings in meningitis include normal studies, ventriculomegaly, ventriculitis, echogenic sulci or convolutional markings, focal and/or diffuse parenchymal echogenic abnormality, extra-axial fluid and encephalomalcia [95].

Ventriculitis manifests as increased echogenecity of the ependymal lining.

MATERIAL AND METHODS

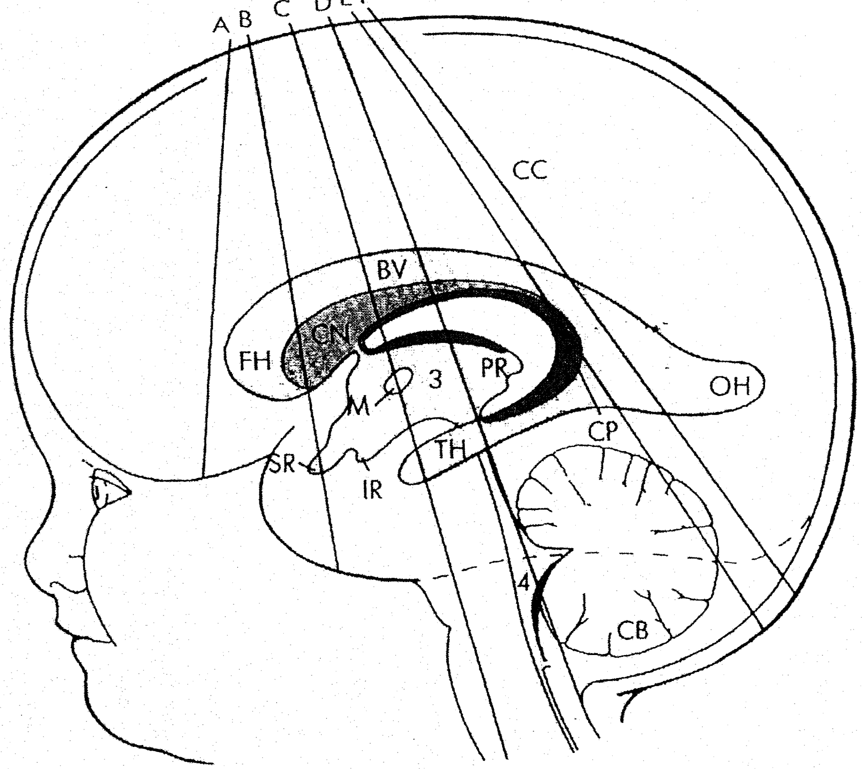
MATERIAL AND METHODS

The present study was carried out in the Department of Radiodiagnosis, MLB Medical College, Jhansi in the duration between Jan. 2001 to Dec. 2002. The patients selected were those neonates who were delivered in this Medical College hospital and those children who were referred here in the outpatients department of Pediatrics and Neonatology; and admitted in neonatal intensive care unit and wards.

A real time cranial sonography was performed on 653 patients. The study comprised of 492 neonates and 161 infants above 1 month of age. 384 out of 492 neonates were premature and 108 full term.

The cut-off value of gestational age for prematurity was taken as 37 weeks or less. Babies with birth weight less than 2500gms were termed low birth weight those less than 1500 termed very low birth weight.

Premature babies formed a major chunk of the study group because the protocol was to routinely screen all premature neonates


A
B
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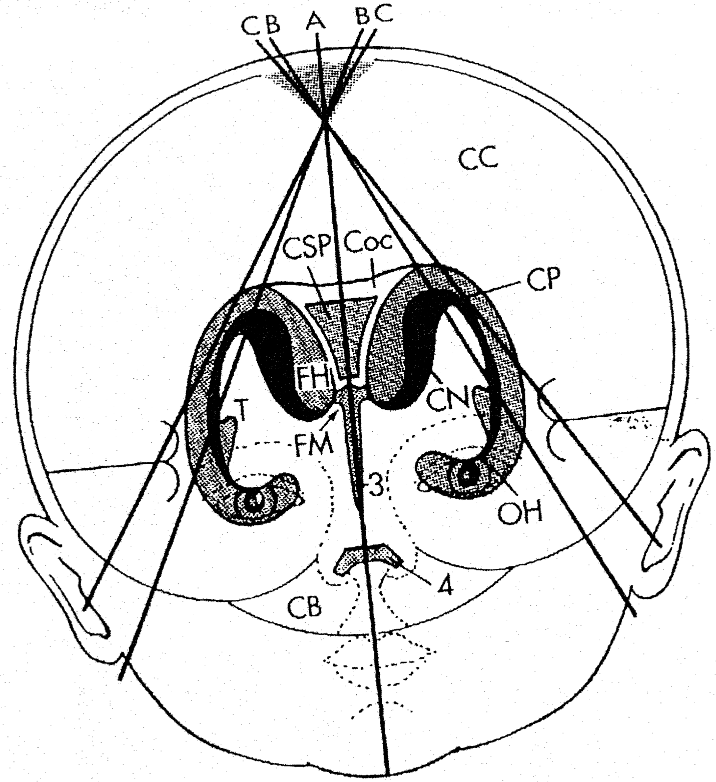
CB

irrespective of clinical status; and to screen full term babies and infants only on appearance of CNS symptoms.

Premature babies were examined as early as possible and scanning was repeated on the 7^{th} and 21^{st} days of the first scan or on development/deterioration of symptoms.

Examination was done by a 3.5 and 5MHz sector probe. Open anterior and posterior fontanelle were used as windows for U/S scanning. Recording was done on gray tone imaging film.

Coronal and sagittal scans were obtained using multiple angulated views. The coronal scan was centered by starting at the trigone so that choroid was symmetric bilaterally. Then multiple slices were taken starting behind the trigone, at the trigone, through the bodies of the lateral ventricles, the frontal horns and lastly as anterior as possible. The sagittal scans were obtained of both lateral ventricles, the midline; and the brain lateral to the ventricles.


CB
A
BC
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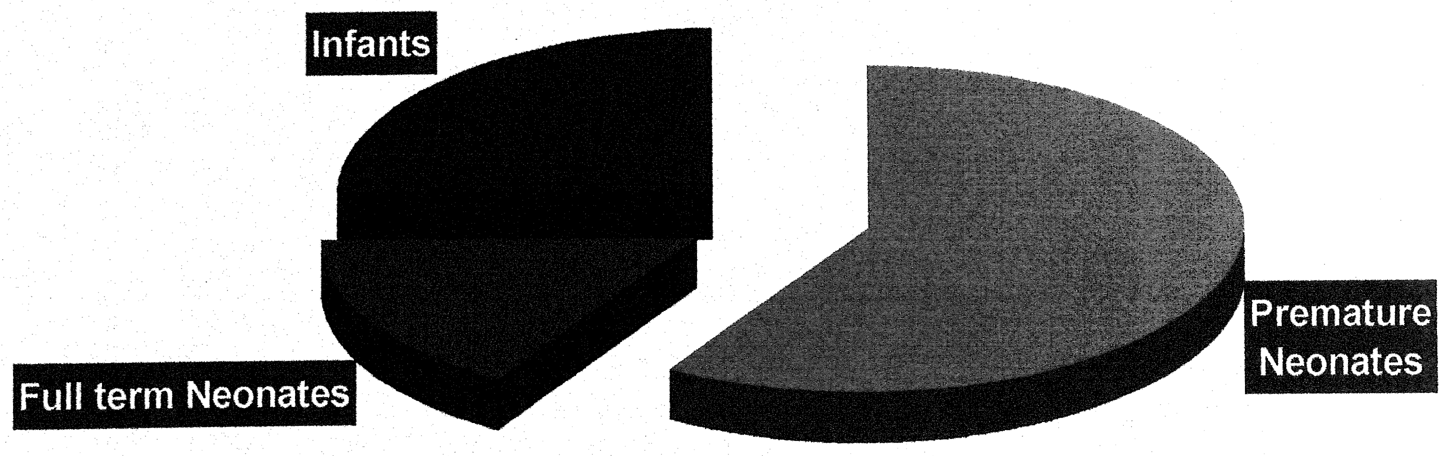
CB

The interpretation of cranial ultrasound was done under the following heads: -

1. Size, shape and echogenicity of ventricles.
2. Periventricular area.
3. LV/HW ratio.
4. Falx cerebri, midline structures and shift thereof.
5. Status of the choroid plexus.
6. Echogenicity of cerebral parenchyma.
7. Sylvian fissure, cingulate and hippocampal gyri.
8. Any abnormal hyper- or hypoechoic area.
9. Any mass lesion.

OBSERVATIONS

DISTRIBUTION OF CASES IN THE STUDY GROUP



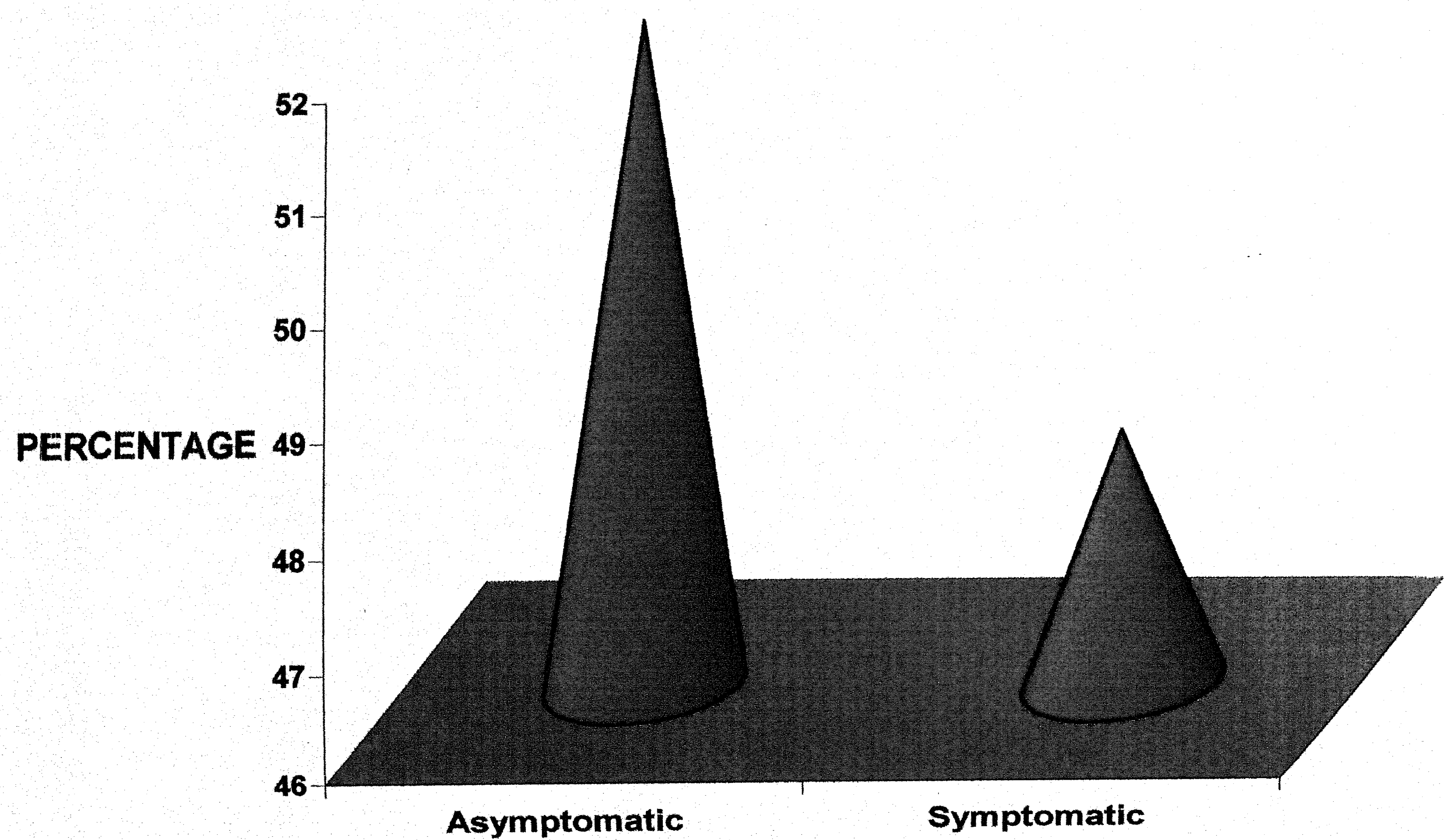
OBSERVATIONS

A real time cranial sonography was performed on 653 patients attending the outpatients department of pediatrics, admitted in the neonatal intensive care units; and wards. The study comprised of 492 neonates and 161 infants above 1 month of age. 384 out of 492 neonates were premature and 108 full term.

Table 1: Distribution of cases according to age

S.No.	AGE RANGE	No. OF CASES	PERCENTAGE
1.	Neonates (Less than 37 weeks)	384	58.80%
2.	Neonates (Full Term)	108	16.54%
3.	Infants (above 1 month of age)	161	24.66%

DISTRIBUTION OF PREMATURE NEONATES

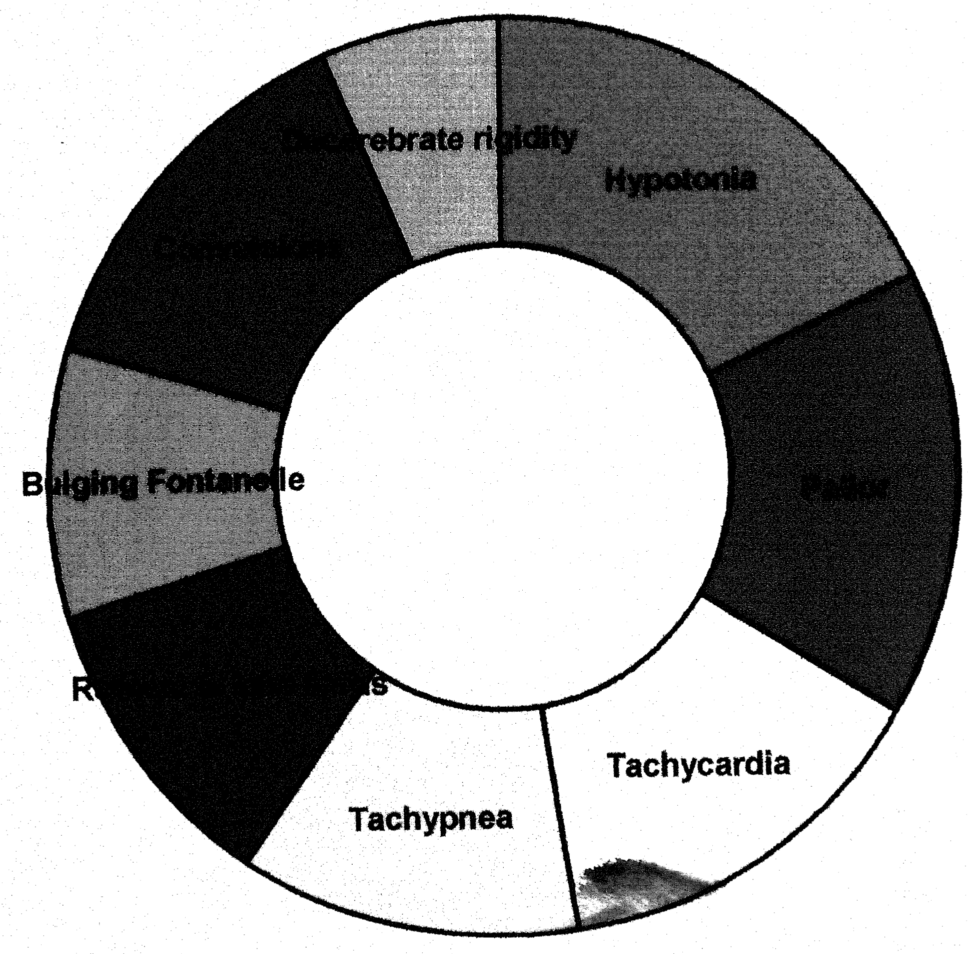


199 out of 384 premature infants were asymptomatic. The remaining 185 prematurely delivered babies had symptoms of pallor, tachycardia, tachypnea, refusal to take feeds, bulging fontanelle, hypotonia, convulsions and decerebrate rigidity.

Table 2: Distribution of premature infants according to symptoms

S.No.		NO. OF CASES	PERCENTAGE
1.	Asymptomatic	199	51.82%
2.	Symptomatic	185	48.18%

SYMPTOMATOLOGY IN PREMATURE NEONATES

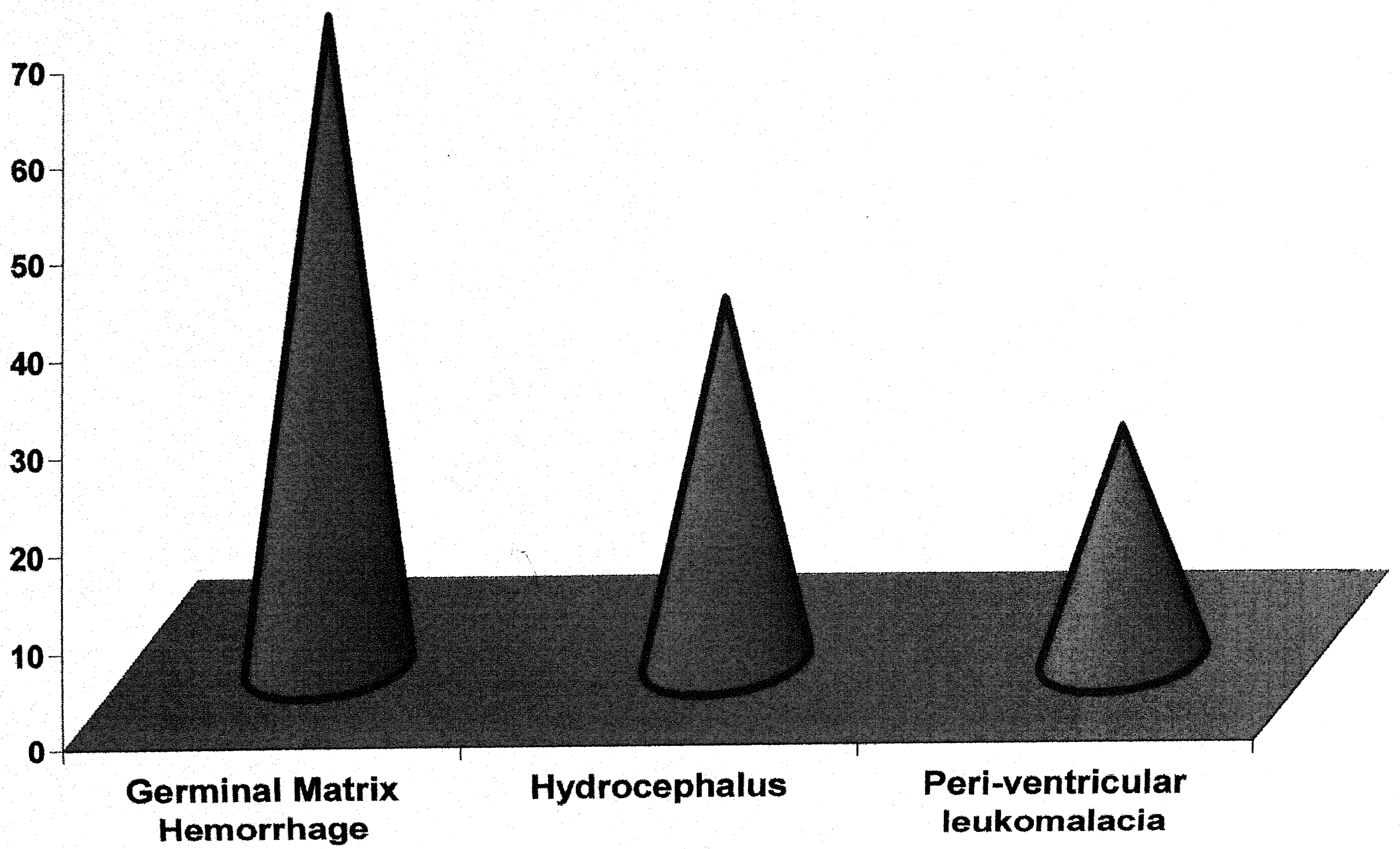


More than one symptom was present at a time in one patient. The most common symptom associated with an abnormal scan in premature babies was hypotonia; closely followed by pallor and tachycardia. The least common was decerebrate rigidity.

Table 3: Symptomatology in premature neonates

S.No.	SYMPTOM	NO. OF CASES	PERCENTAGE
1.	Hypotonia	123	66.48%
2.	Pallor	107	57.83%
3.	Tachycardia	96	51.89%
4.	Tachypnea	84	45.40%
5.	Refusal to take feeds	72	38.91%
6.	Bulging Fontanelle	65	35.13%
7.	Convulsions	96	51.89%
8.	Decerebrate Rigidity	43	23.24%

SONOGRAPHIC ABNORMALITIES IN SYMPTOMATIC PREMATURE BABIES

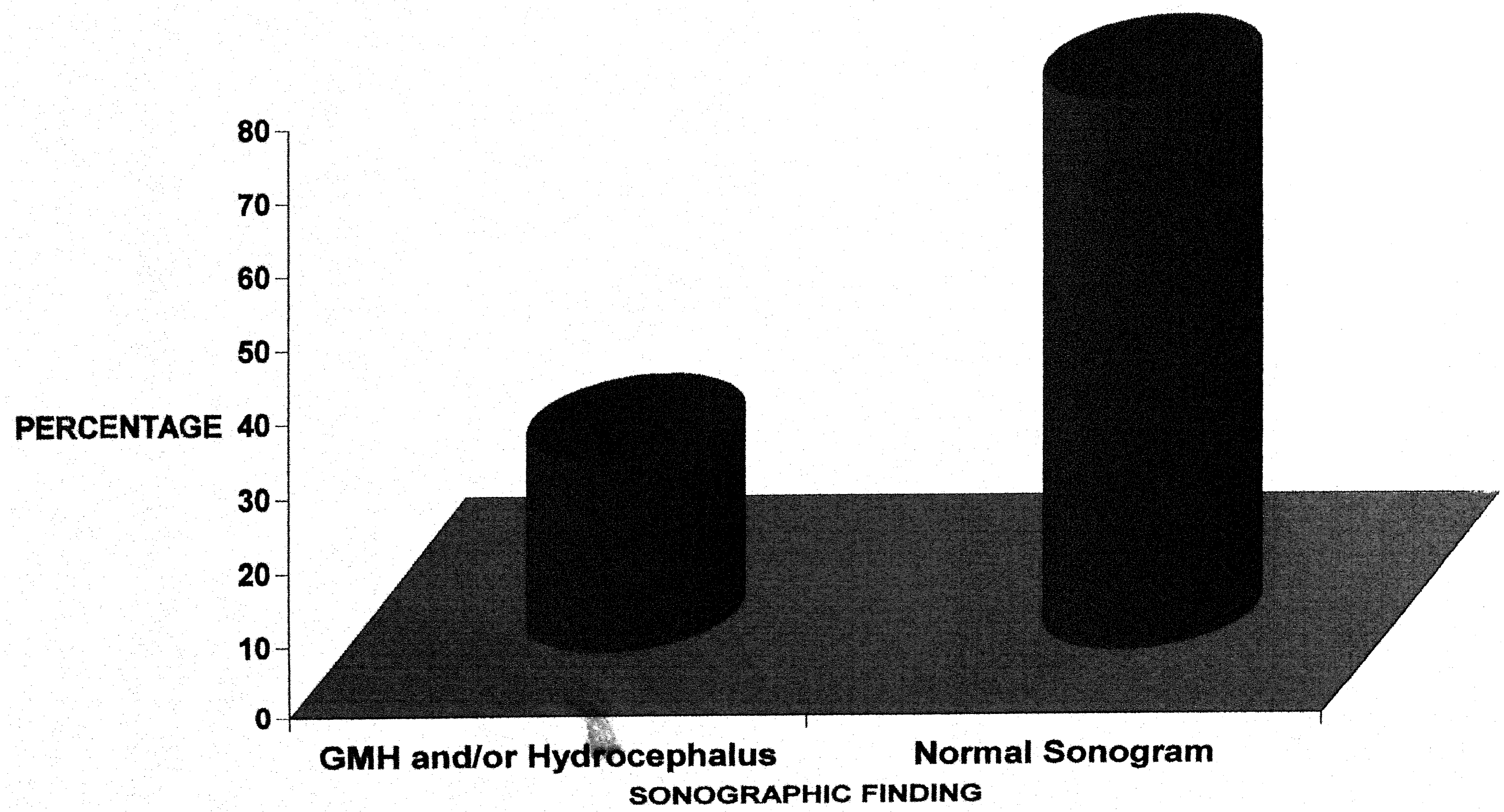


Germinal matrix hemorrhage was the commonest abnormality (67.02%) found in premature babies. The other abnormalities found were; hydrocephalus and periventricular leukomalacia. Hydrocephalus was a common accompaniment of intraventricular hemorrhage but was also observed in otherwise normal patients.

Table 4: Range of sonographic abnormalities in symptomatic premature babies.

S.No.	SONOGRAPHIC ABNORMALITY	NO.OF CASES	PERCENTAGE
1.	Germinal Matrix hemorrhage	124	67.02%
2.	Hydrocephalus	69	37.29%
3.	Peri-ventricular Leukomalacia	44	23.78%

SONOGRAPHIC FINDINGS IN ASYMPTOMATIC PREMATURE BABIES

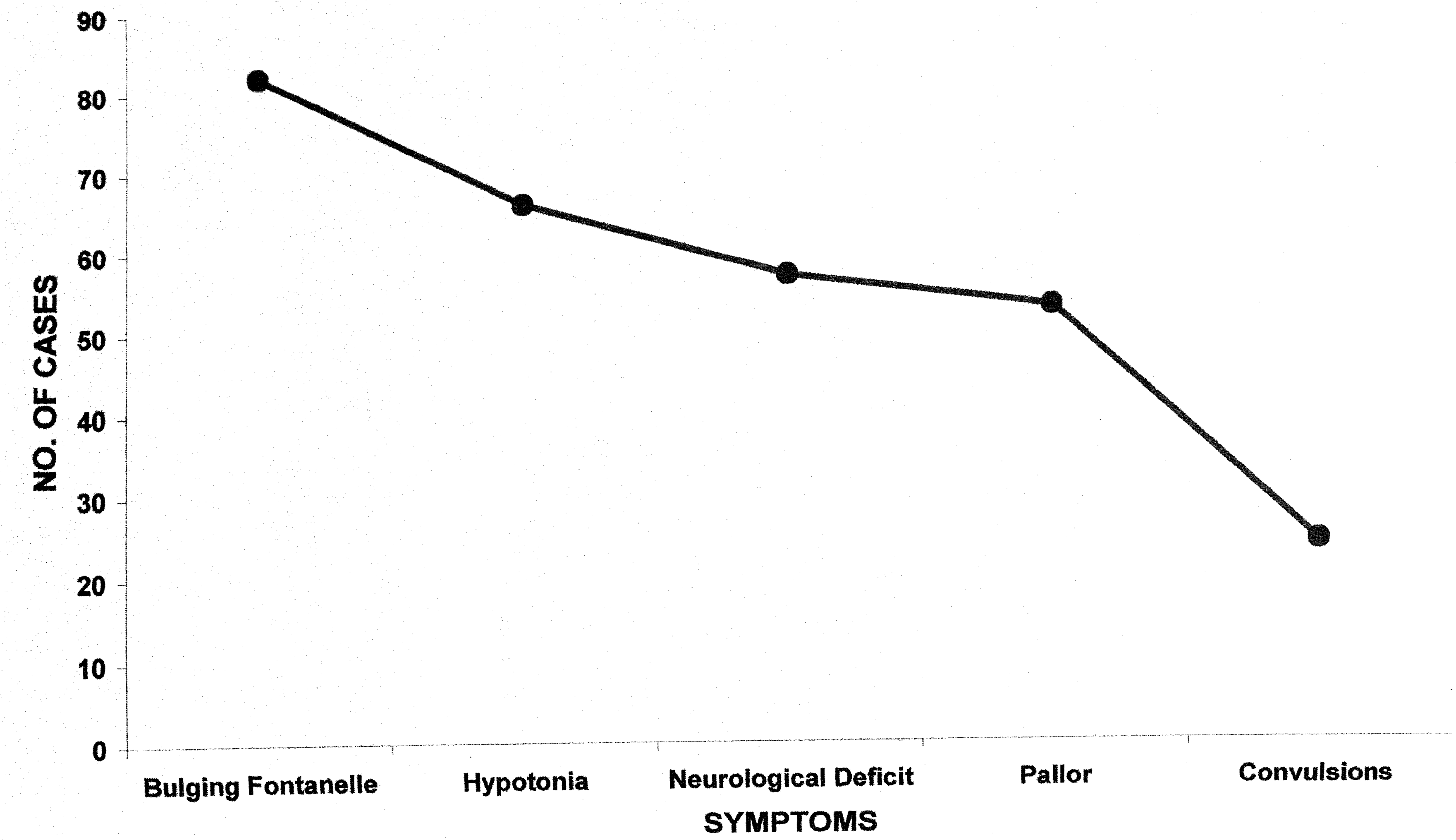


In 199 asymptomatic premature babies, cranial sonography was abnormal in 51 (25.62%) cases. The pathologies seen were germinal matrix hemorrhage and hydrocephalus.

Table 5: Sonographic findings in asymptomatic premature babies

S.No.	SONOGRAPHIC FINDING	No. OF CASES	PERCENTAGE
1.	Germinal matrix Hemorrhage and/or Hydrocephalus	51	25.63%
2.	Normal sonogram	148	74.37%

RANGE OF CLINICAL FEATURES IN FULL TERM NEONATES

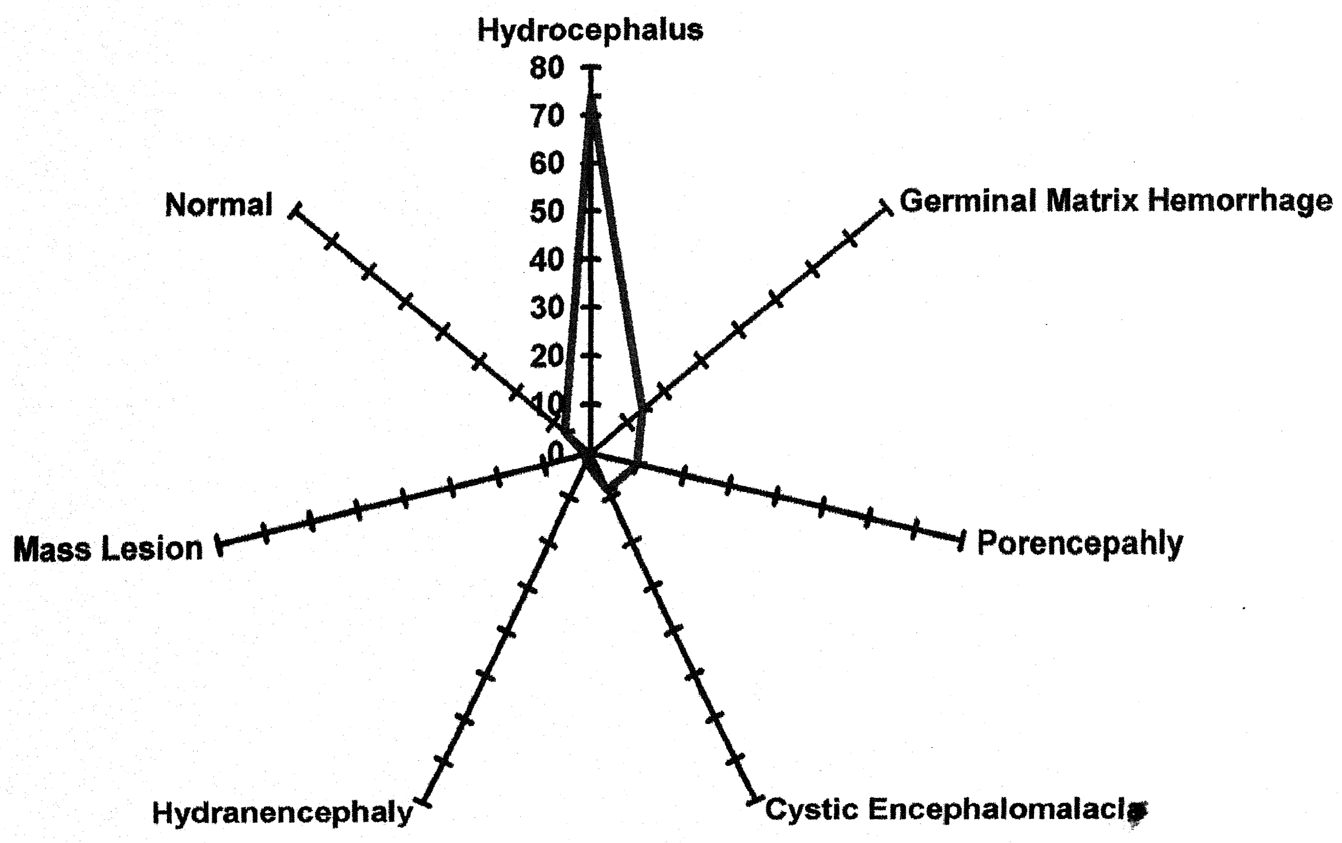


The commonest feature among the 108 full term neonates was bulging fontanelle.

Table 6: Range of Clinical features in 108 full term neonates

S.No.	SYMPTOM	NO.OF CASES	PERCENTAGE
1.	Bulging Fontanelle	82	75.92%
2.	Hypotonia	66	61.11%
3.	Neurological Deficit	57	52.77%
4.	Pallor	53	49.07%
5.	Convulsions	24	22.22%

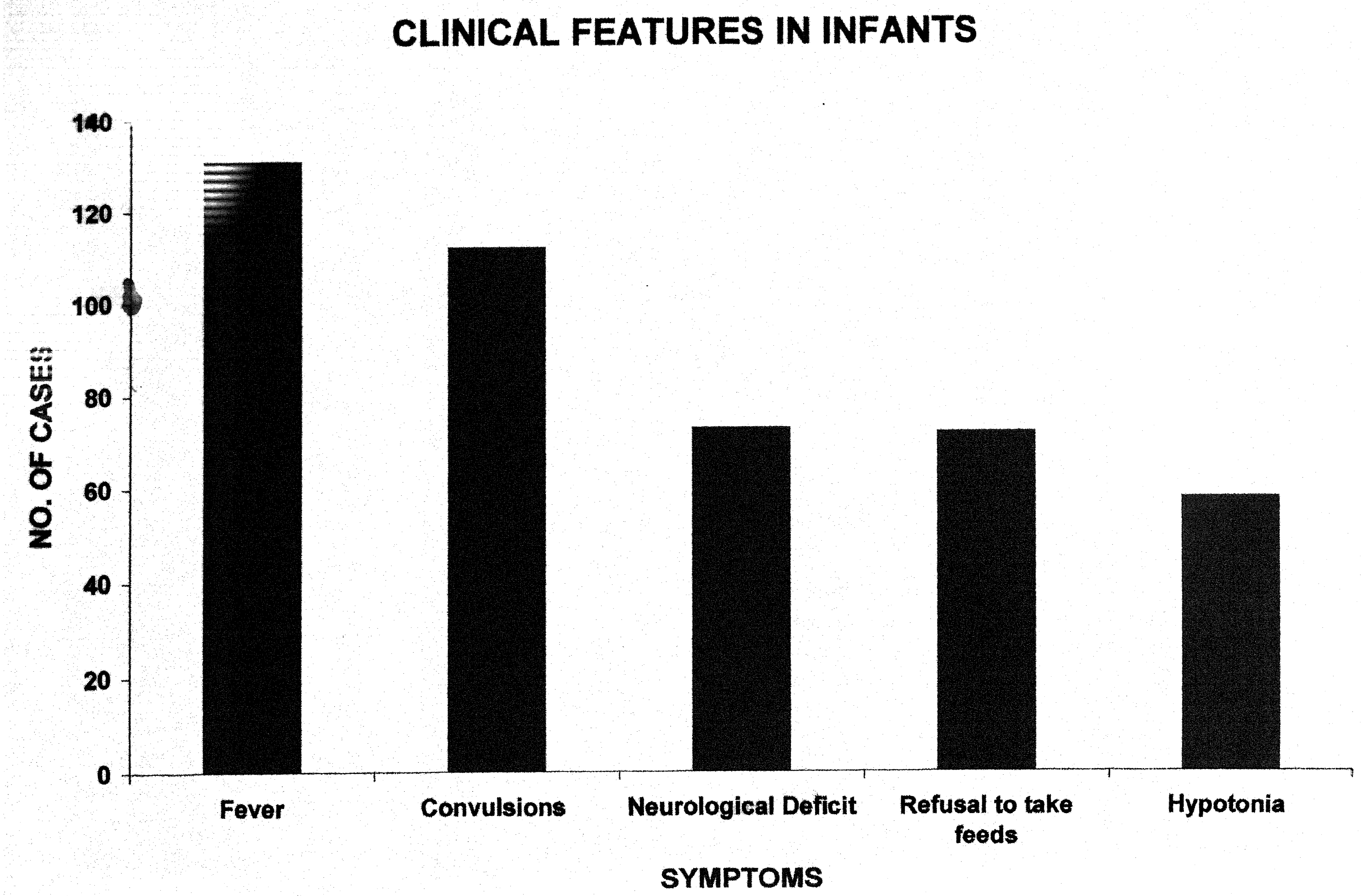
SONOGRAPHIC ABNORMALITIES IN FULL TERM NEONATES



Majority of these patients showed hydrocephalus. The other detected abnormalities were porencephalic cysts, germinal matrix hemorrhage, hydranencephaly and a mass lesion. No abnormality was found in 7 patients.

Table 7: Sonographic abnormalities in 108 full term neonates

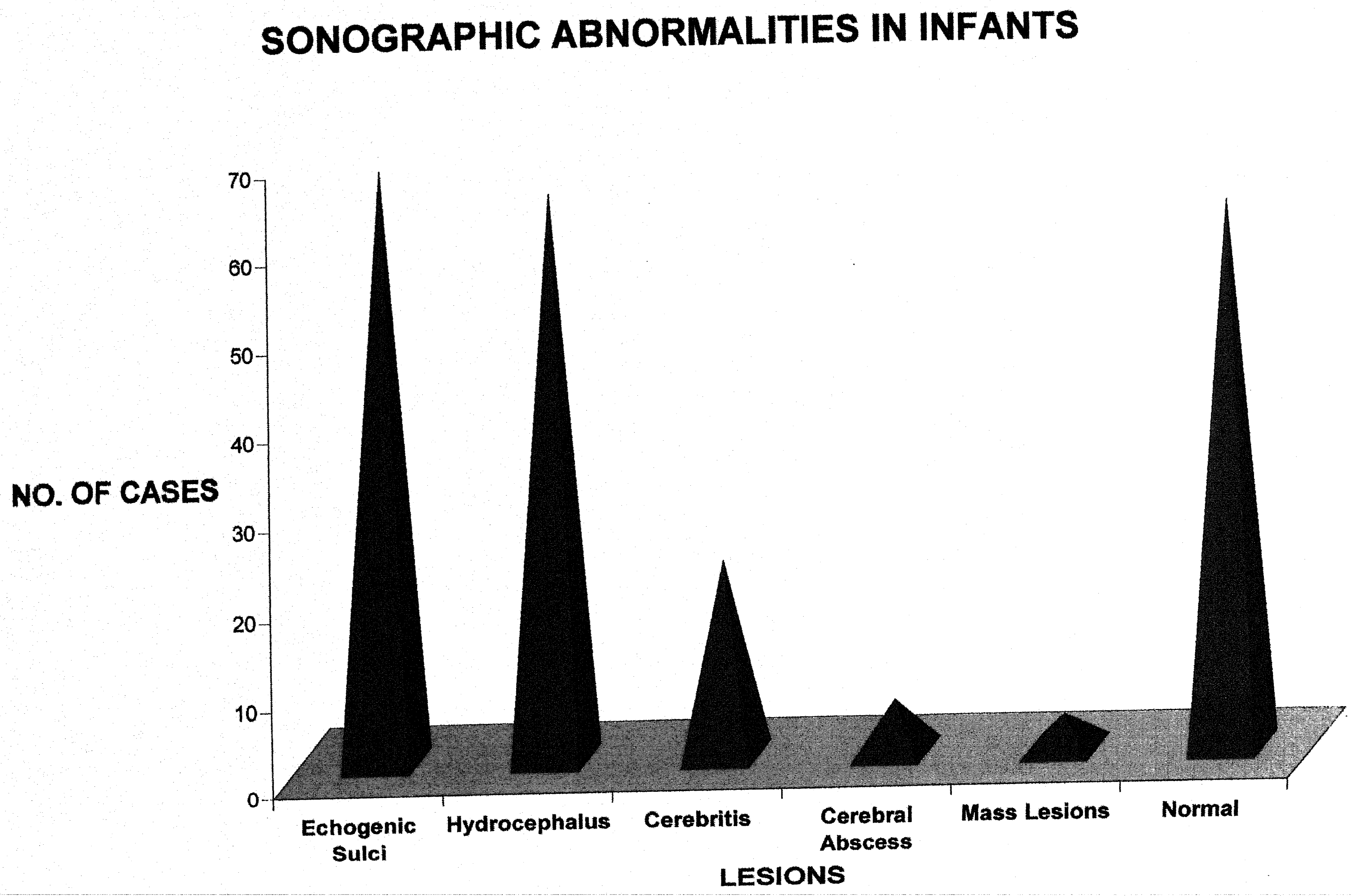
. No.	SONOGRAPHIC ABNORMALITY	NO.OF CASES	PERCENTAGE
	Hydrocephalus	74	68.51%
	Germinal Matrix Hemorrhage	14	12.97%
	Porencephaly	10	9.25%
	Cystic Encephalomalacia	8	7.40%
	Hydranencephaly	2	1.86%
	Mass Lesion	1	0.93%
	Normal	7	6.48%


CLINICAL FEATURES IN INFANTS
NO. OF CASES
140
120
100
80
60
40
20
0
Fever
Convulsions
Neurological Deficit
Refusal to take feeds
Hypotonia
SYMPTOMS

Convulsions were the most common neurological feature for the request of a cranial sonography in the 161 infants above 1 month of age in the study group.

Table 8: Clinical features in 161 infants above 1 month of age

S.No.	SYMPTOM	NO.OF CASES	PERCENTAGE
1.	Fever	131	81.36%
2.	Convulsions	112	69.56%
3.	Neurological Deficit	73	45.34%
4.	Refusal to take feeds	72	44.72%
5.	Hypotonia	58	36.02%


SONOGRAPHIC ABNORMALITIES IN INFANTS
NO. OF CASES
70
60
50
40
30
20
10
0
Echogenic Sulci
Hydrocephalus
Cerebritis
Cerebral Abscess
Mass Lesions
Normal
LESIONS

Echogenic sulci with ventriculitis with/without hydrocephalus were most commonly found in these patients. The other abnormalities were cerebritis, cerebral abscess and mass lesions.

Table 9: Sonographic abnormalities in 161 infants

S.No.	SONOGRAPHIC ABNORMALITY	NO OF CASES	PERCENTAGE
1.	Echogenic Sulci	67	41.61%
2.	Hydrocephalus	64	39.75%
3.	Cerebritis	22	13.66%
4.	Cerebral Abscess	6	3.72%
5.	Mass Lesions	4	2.48%
6.	Normal	62	38.50%

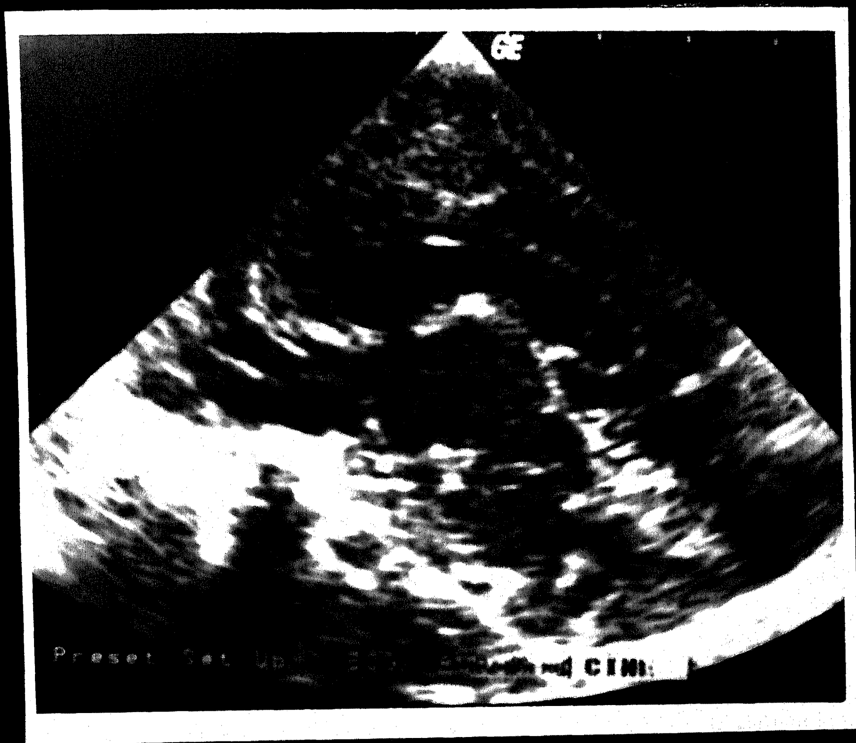


Fig. 1 Echogenic focus in the Caudothalamic Groove: Germinal Matrix Hemorrhage Grade I

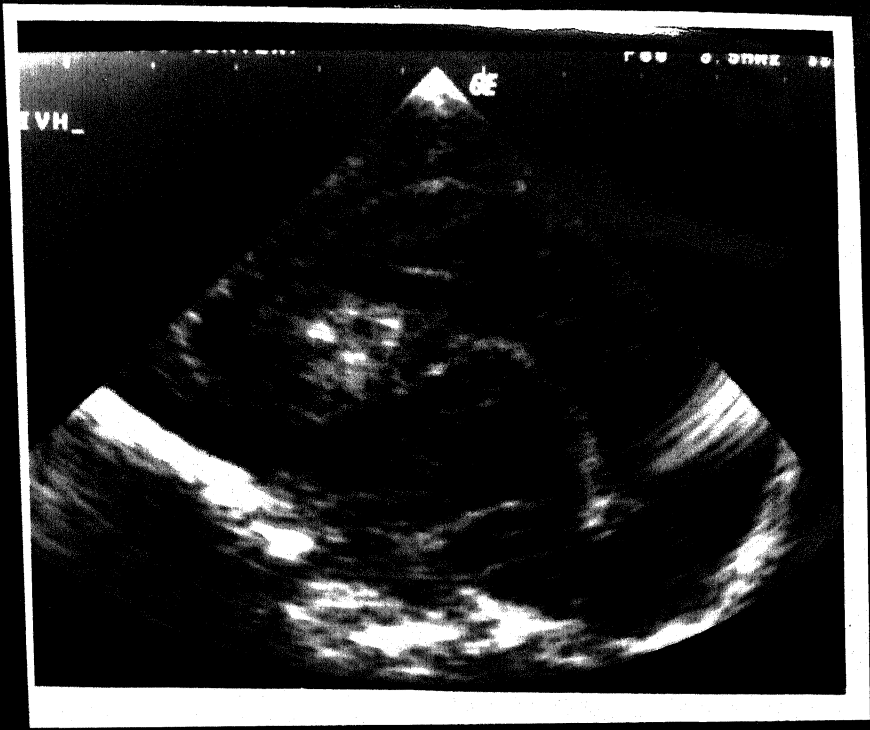


Fig. 2 Echogenic blood in lateral ventricle without ventricular dilatation: Germinal Matrix Hemorrhage Grade II

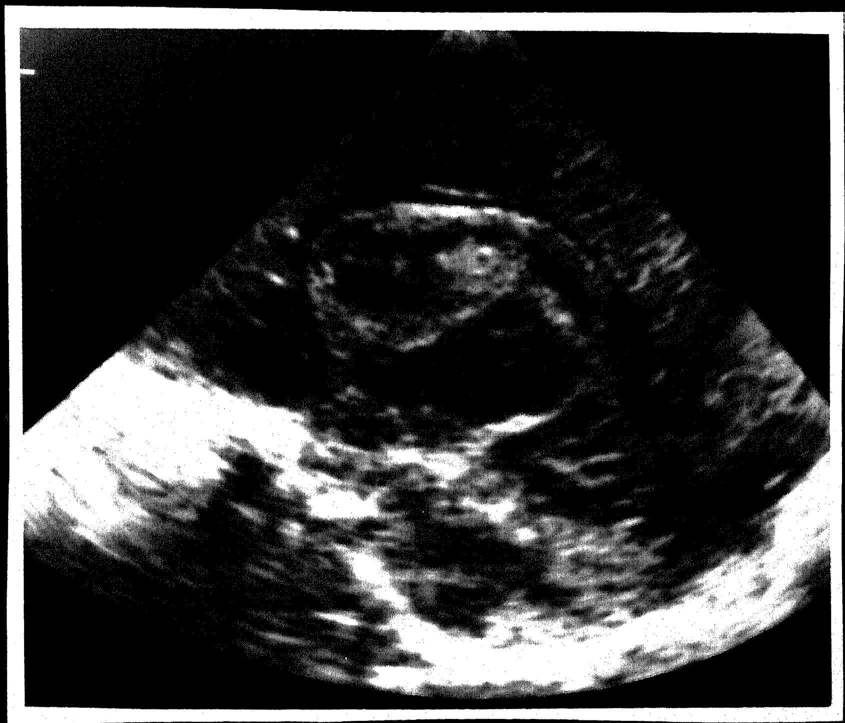


Fig. 3 Bulky Choroid without Ventricular dilatation suggestive of Grade II GMH

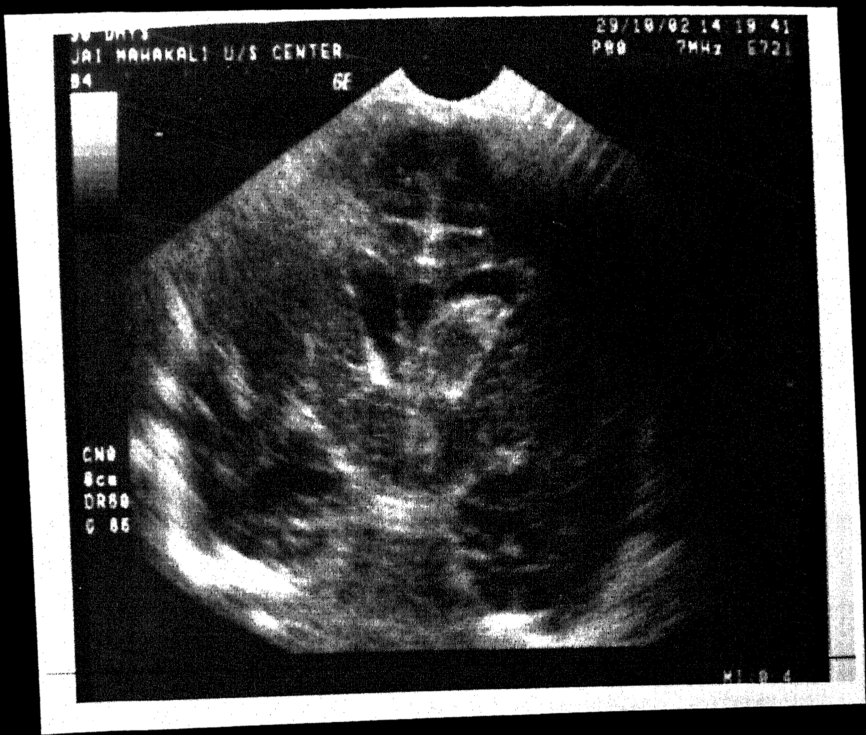


Fig. 4 Echogenic blood in lateral ventricle on left side with ventricular dilatation: Left sided Grade III GMH. Grade I GMH on right.

Fig. 5 Extension of hemorrhage into adjacent brain parenchyma:
GMH Grade IV.

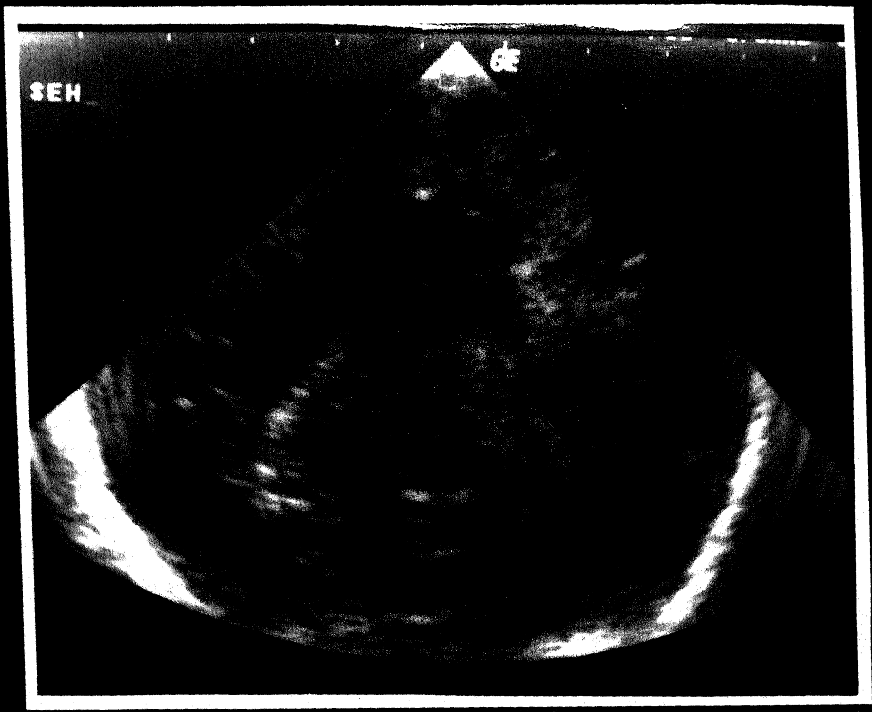


Fig. 6 Increased echogenicity just lateral and superior to lateral ventricle on left side: Periventricular Leukomalacia

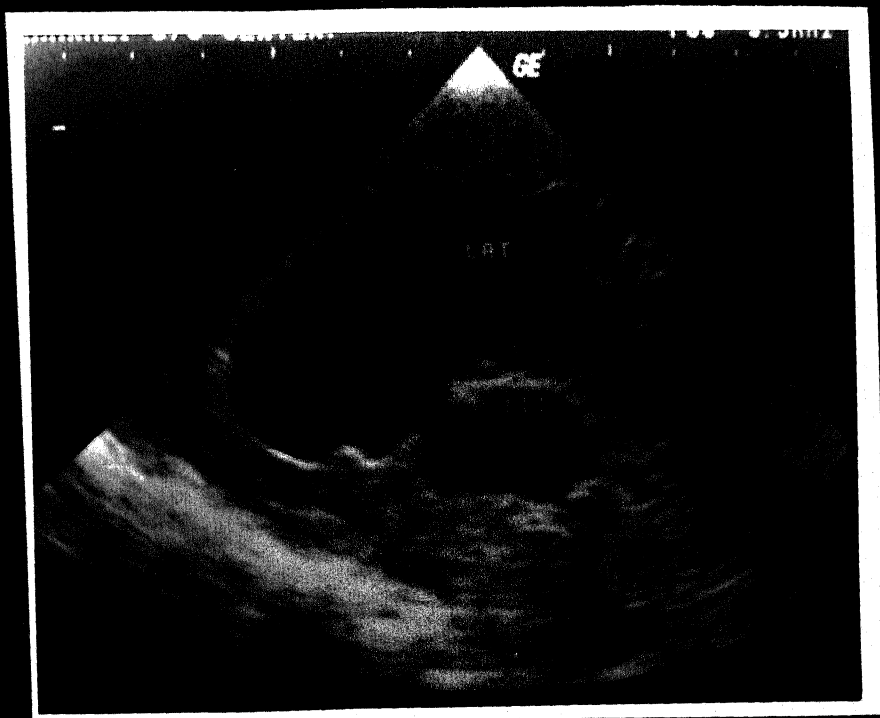


Fig. 7 Sagittal sonogram in a case of Hydrocephalus showing Foramen of Monroe

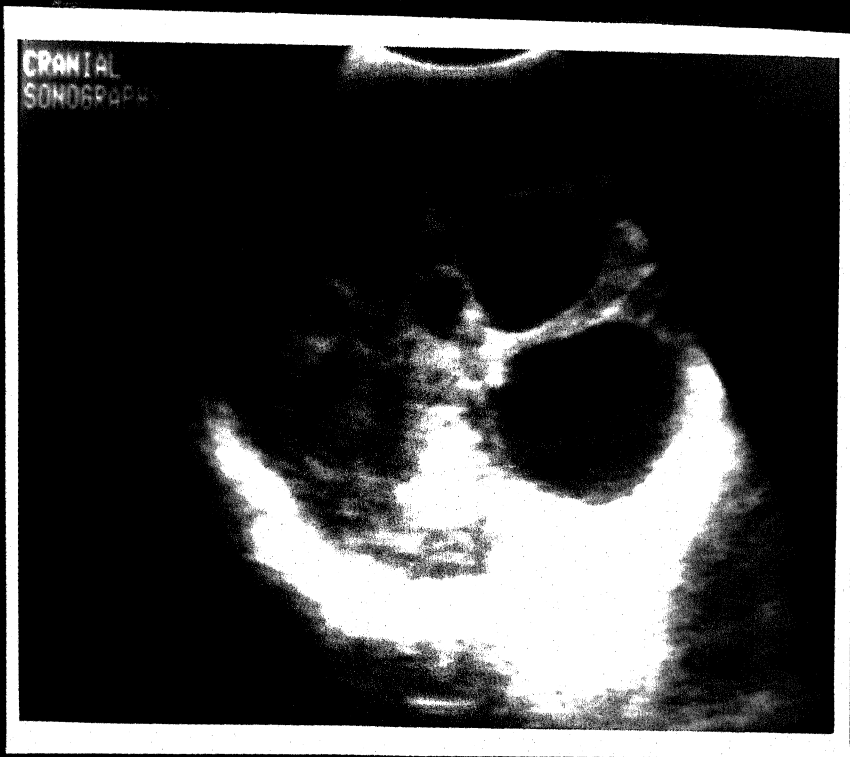


Fig. 8 Coronal sonogram showing dilated lateral and third ventricles: Aqueductal Stenosis

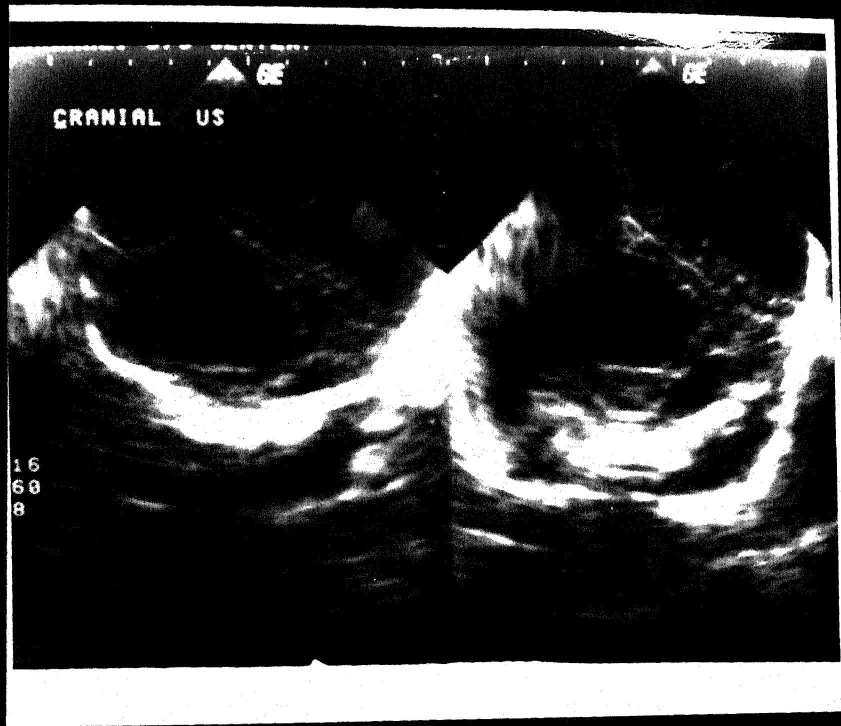


Fig. 9 Dilated lateral and third ventricles: Aqueductal Stenosis

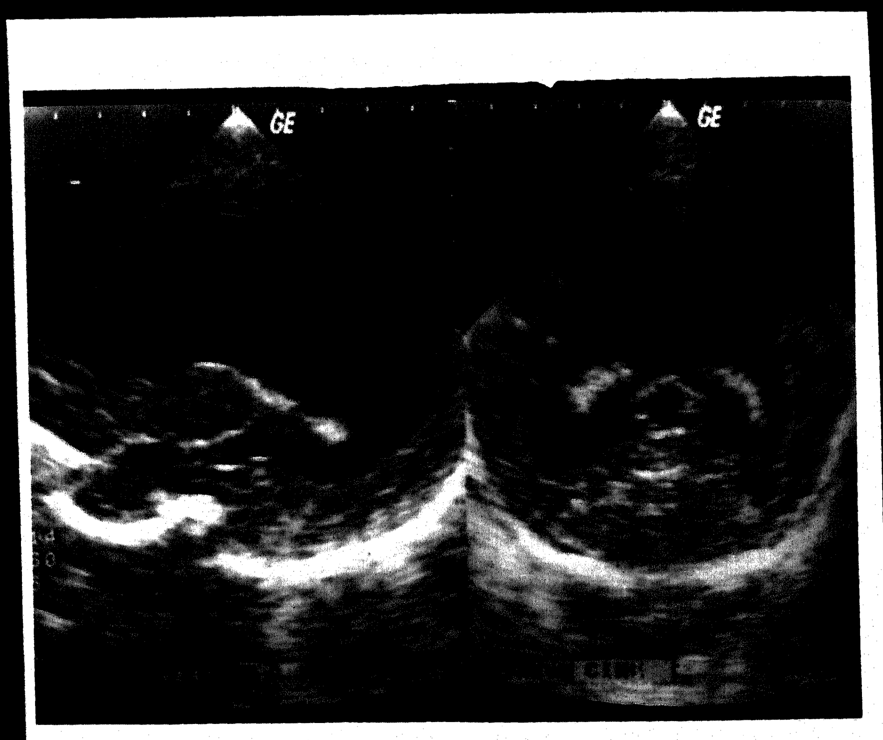


Fig. 10 Dilated lateral ventricles: Obstruction at Foramen of Monroe

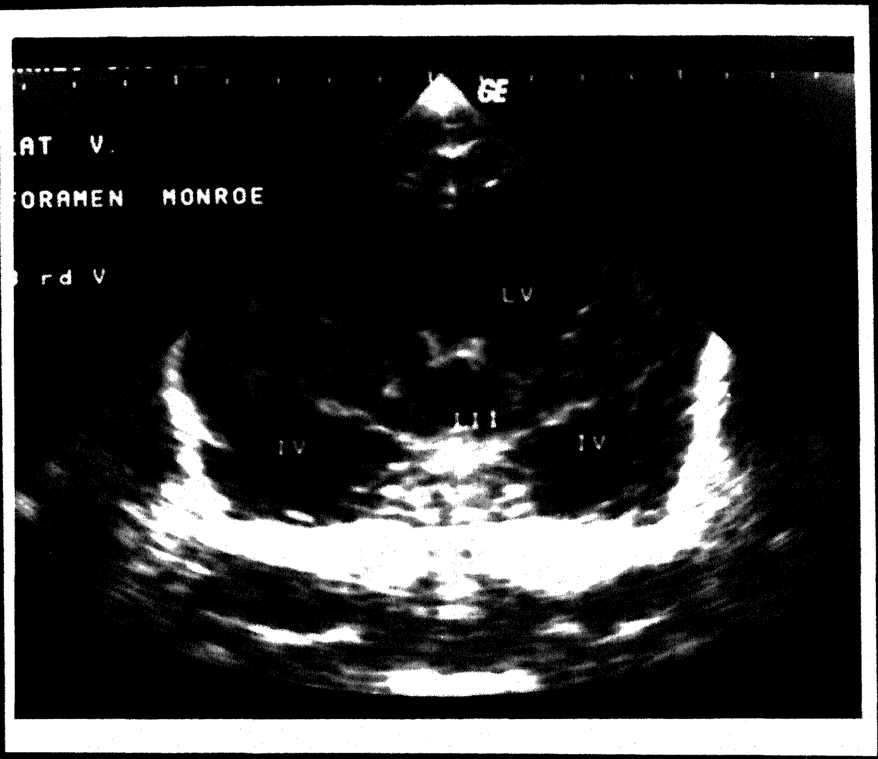


Fig. 11 Aqueductal Stenosis

Fig. 12 Sagittal sonogram in a case of Hydrocephalus

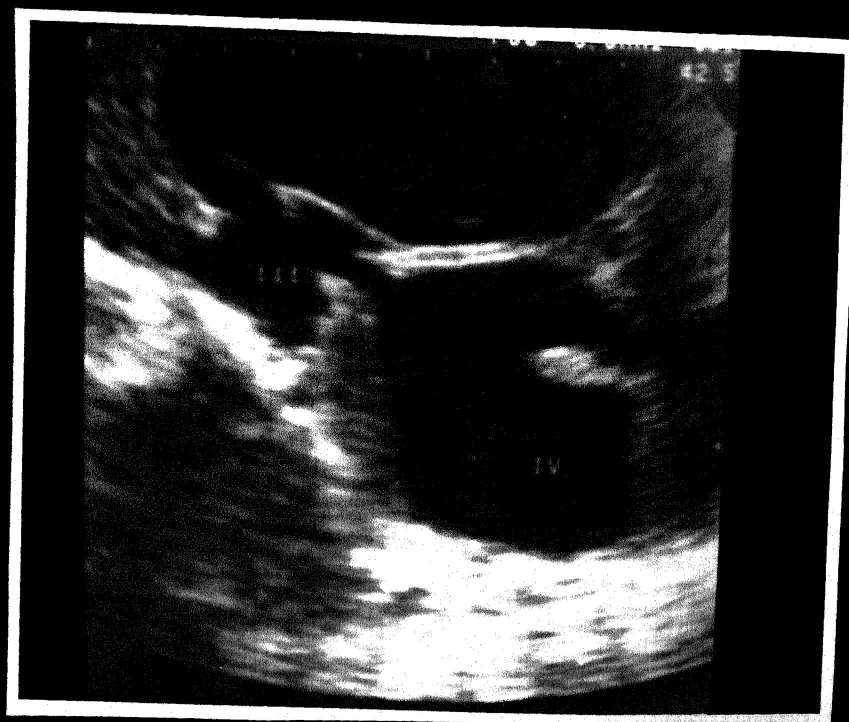


Fig. 13 Sagittal sonogram showing dilatation of all four ventricles. Foramen of Monroe and Aqueduct of Sylvius are beautifully demonstrated.

Fig. 14 Cystic area in left Fronto-Parietal region communicating with ventricle: Porencephaly

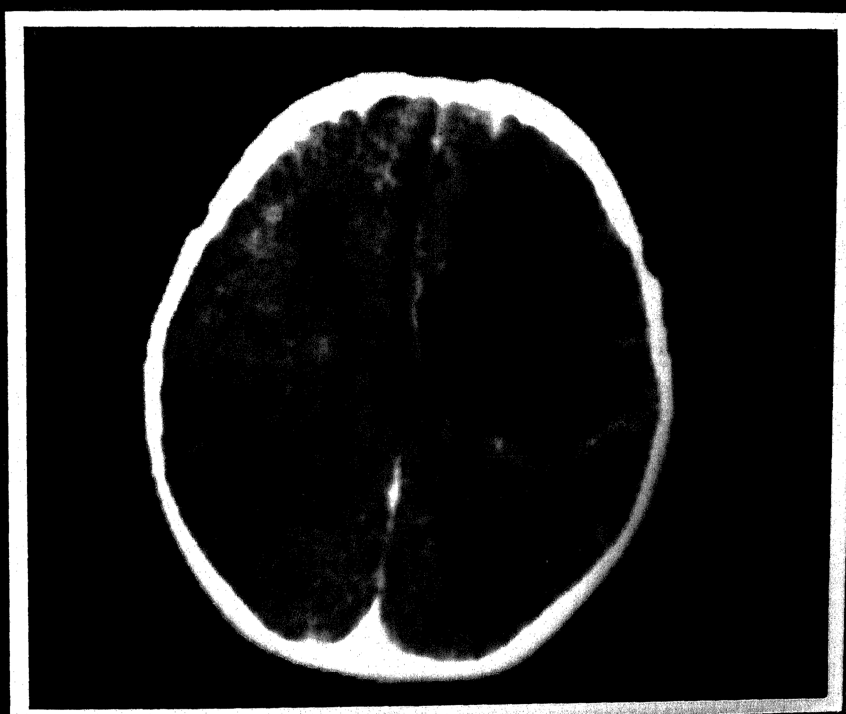


Fig. 15 Axial CT section of the above case confirming the diagnosis.

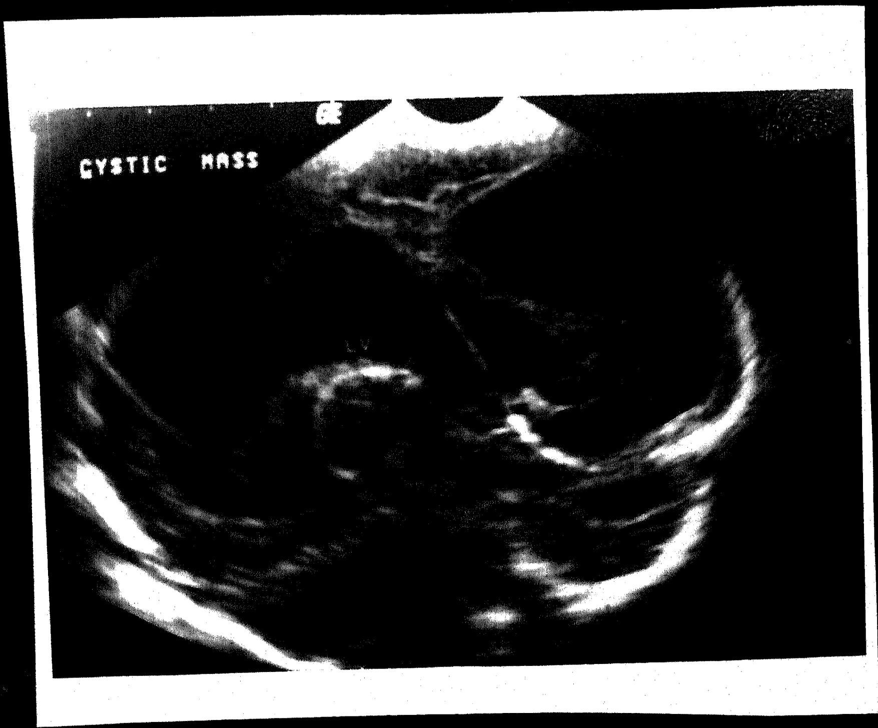


Fig. 16 Large cystic area with septae in left Fronto-Parietal region on Coronal Cranial sonogram: Porencephalic Cyst

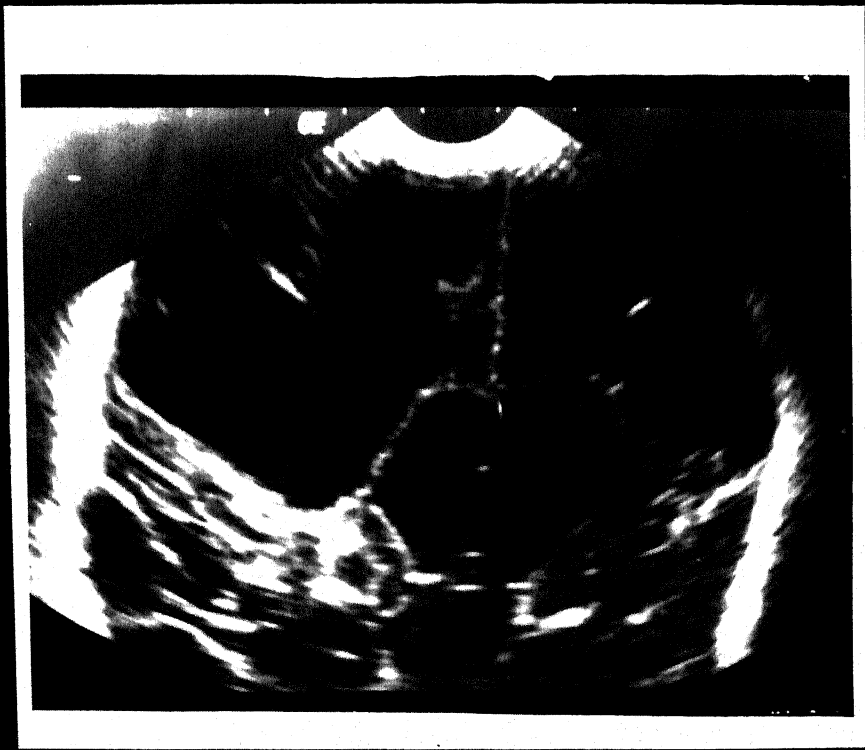


Fig. 17 Bilateral multiple cystic areas distributed throughout the brain parenchyma: Cystic Encepahlomalacia.

Fig. 18 Sagittal Scan of the above case

Fig. 19 Coronal Cranial Sonogram without any appreciable brain parenchyma: Hydranencephaly

Fig. 20 Cranial sonogram through mastoid fontanelle showing falx and only posterior part of brain parenchyma: Hydranencephaly.

Fig. 21 Mass in left Fronto-Parietal region.

Fig. 22 Solid echogenic mass in right Parietal region.

Fig. 23 Right Parietal mass with Hydrocephalus

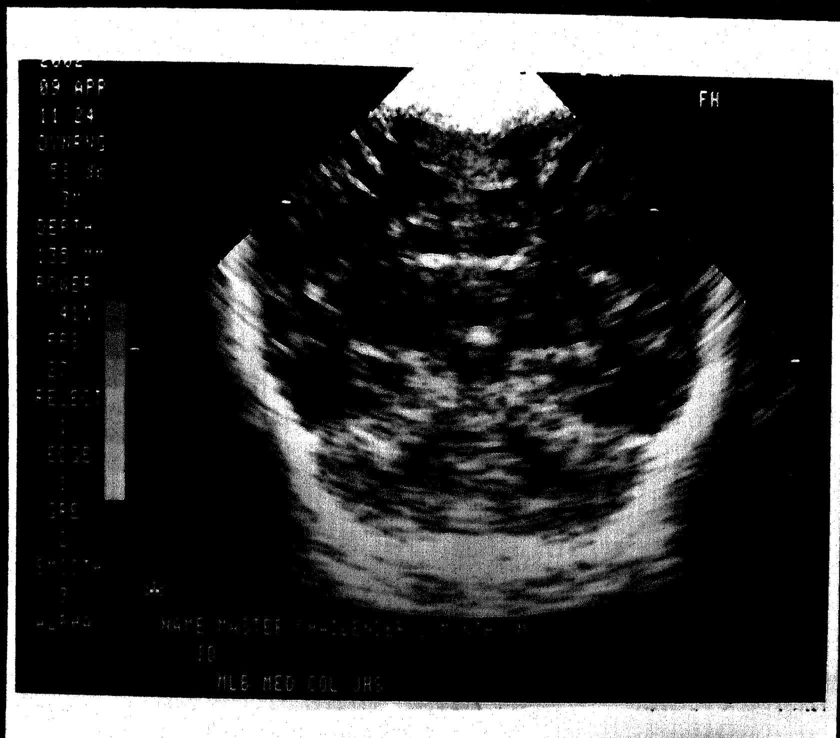


Fig. 24 Echogenic Sulci and Gyri with periventricular haziness: Cerebritis

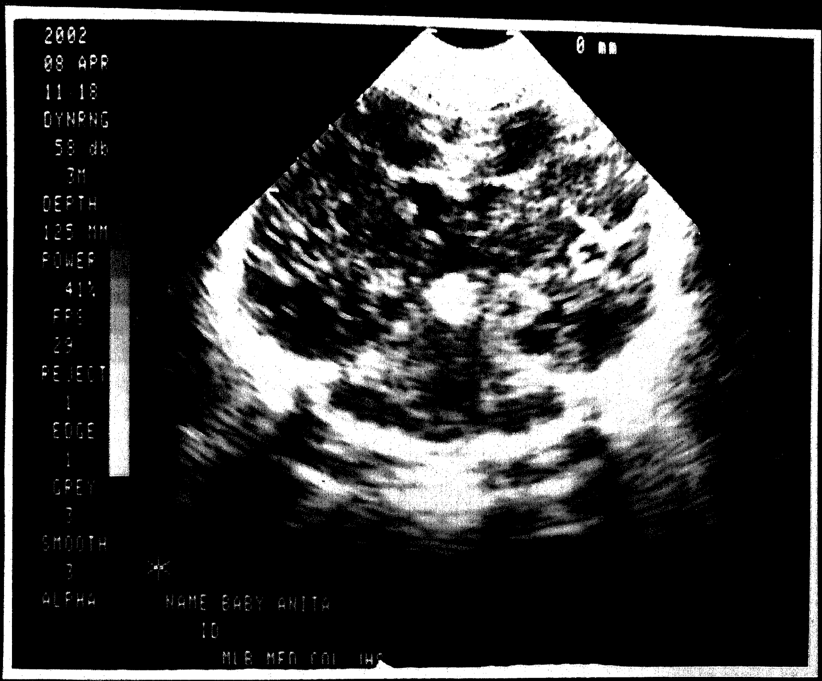


Fig. 25 Echogenic Sulci and Gyri with ventricular dilatation: Meningitis

DISCUSSION

DISCUSSION

Transfontanelle cranial sonography has an important place in the evaluation of the neurological status of neonates and infants, whether symptomatic or asymptomatic.

A total of 653 patients were subjected to cranial sonography out of which 384 were premature. The commonest symptom among the symptomatic premature babies was hypotonia.

This finding is in accordance with the study by Mercuri E et al (1998) who evaluated cranial sonography and neurological examination in a cohort of infants. They found that out of 43 patients showing deviant neurological pattern, 25 had an abnormal tone [96].

In our study the commonest finding in premature babies was germinal matrix hemorrhage. This is consistent with the findings of Sims ME et al (1986) who found that out of 170 positive cranial ultrasound scans in premature infants, intracranial hemorrhage was detected in 150 patients [97].

This finding is also supported by Allan WC et al in their review article on neonatal cerebral pathology diagnosed by ultrasound. They observed that one third to one half of all premature infants had a periventricular-intraventricular hemorrhage [98].

Kirks DR et al also found a high incidence (44%) of intracranial hemorrhage in premature neonates [99].

Vohr B and Ment LR in an article on intraventricular hemorrhage in preterm infants suggested that IVH is a common finding and the incidence of IVH ranges from 40 to 60% [16].

Our study also revealed that the incidence of germinal matrix hemorrhage fell as more and more babies reached full term. The incidence of GMH in full term infants in our study was 12.97% as against the high incidence of 67.02% in premature infants.

These findings are similar to the ones noted by Perry RN et al who observed that there was a reduction in the incidence of cerebroventricular hemorrhage with increasing gestation [100].

Hydrocephalus was a common accompaniment of intraventricular hemorrhage in the present study. This is in accordance with various studies on subependymal and intraventricular hemorrhage, most of which mention an association of hydrocephalus with subependymal/ intraventricular hemorrhage [1, 17, 63,101].

Kirks DR et al (1986) observed that hydrocephalus in periventricular/intraventricular hemorrhage can be acute or may manifest as a late complication [102].

Acute dilatation of the ventricles in PVH/IVH may be non-obstructive; due to blood in the ventricles. Chronic dilatation of ventricles developing on a background of PVH/IVH is due to obstruction to CSF flow secondary to reactive gliosis. This inflammatory process is found either over the convexities of the cerebral hemispheres with occlusion of the arachnoid villi or in the posterior fossa with obstruction of outflow of the fourth ventricle. The obstruction may also occur at the Aqueduct of Sylvius by an acute clot or reactive gliosis.

The association of hydrocephalus with PVH/IVH was also observed by Perry RN et al [100] who reported ventricular dilatation in 20 of the

33 infants of less than 32 weeks gestation with hemorrhage and 7 of the 13 infants of 32 weeks or more; with hemorrhage.

Sims et al (1986) also discovered that post-hemorrhagic ventriculomegaly occurred in approximately one half of premature infants developing PVH/IVH.

In the present study 25.62% premature infants had germinal matrix hemorrhage without manifesting any signs thereof. This means that the usage of clinical criteria to screen infants for GMH would lead to underdiagnosis.

This finding is supported by the study by Paul DA et al (1999) who applied clinical screening criteria for detection of IVH. They found that these criteria had a sensitivity of 51%, a specificity of 62% and a positive predictive value of only 31%. They noted that selective screening using clinical risk factors would have missed 49% cases of IVH including 46% grade III and IV hemorrhage [103].

In the study by Perry et al [100] none of the 33 infants less than 32 weeks gestation had signs of hemorrhage.

Hydrocephalus was the most common sonographic finding in the 108 full term neonates in our study. Edwards et al (1981) in their study on 56 neonates also found ventricular enlargement to be the most common finding. They diagnosed ventriculomegaly in 21 out of 56 cases [50].

The most common neurological complaint in the for which a cranial sonography was requested in the 161 infants above 1 month of age in the present study was convulsions. The most common symptom however was fever. 62 (38.50%) patients in this group had a normal sonographic study. The accompaniment of fever with convulsions suggests that such patients might be suffering from febrile convulsions. 67 of these patients were found to have findings in favour of meningitis and/or cerebritis.

Data were not found to support this finding probably because literature on cranial sonography is largely available in western setting where infectious diseases are not very common.

Hydrocephalus was a common manifestation in patients suffering meningitis. This can again be attributed to inflammatory arachnoiditis and secondary obstruction to CSF flow.

CONCLUSION

CONCLUSIONS

The present work has been done to evaluate the role of cranial sonography in neonates and infants. The study specially stressed on its role in detecting cerebral lesions in prematurely born infants.

The results are summarized as follows:-

1. Cranial sonography is a sensitive and specific method to detect cerebral pathologies. Germinal matrix hemorrhage, ventricular dilatation, lesions in the parenchyma and ventricles are easily diagnosed.
2. Germinal matrix hemorrhage is the most common lesion detected in prematurely born infants.
3. Hypotonia is the most common manifestation of germinal matrix hemorrhage.
4. A large number of premature infants with subependymal/intraventricular hemorrhage are asymptomatic.
5. Allocation of premature infants for cranial sonography on the basis of clinical criteria would miss a substantial number of patients with intracranial abnormalities.

6. Ventricular dilatation is a common accompaniment of subependymal/intraventricular hemorrhage.
7. Hydrocephalus is the most common finding in full term neonates.
8. Hydrocephalus in full term neonates most commonly presents with bulging fontanelle.
9. The most common lesion detected in infants above 1 month of age was meningitis.
10. The commonest presenting feature in meningitis was convulsions.
11. Cranial sonography is a mandatory investigation in all infants born prematurely.
12. Cranial sonography is an innocuous, repeatable and easy method to detect intracranial lesions in neonates and infants which can be used safely to diagnose and follow patients with cerebral pathologies.
13. Because of being safe, non-ionizing and easily available; cranial sonography can be used as a primary imaging modality to detect intracerebral lesions in children with open anterior fontanelle.

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S.No	Name	Gestational Age	Sex	Clinical Presentation	Sonographic Finding
1	B.Abhilasha	33 weeks	F	Asymptomatic	GMH. Hydrocephalus
2	B.Abhishek	33 weeks	M	Hypotonia, Pallor. Tachycardia, Tachypnoea	GMH. Hydrocephalus
3	B.Abhishek	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH. Hydrocephalus
4	B.Adil	36 weeks	M	Asymptomatic	WNL
5	B.Afaque	37 weeks	M	Asymptomatic	WNL
6	B.Ajay	33 weeks	M	Decerebrate Rigidity. Refusal to take feeds	PVL
7	B.Ajvendra	36 weeks	M	Asymptomatic	GMH, Hydrocephalus
8	B.Alisha	36 weeks	F	Asymptomatic	WNL
9	B.Alka	36 weeks	M	Asymptomatic	WNL
10	B.Amantri	36 weeks	F	Asymptomatic	WNL
11	B.Ami	36 weeks	M	Asymptomatic	WNL
12	B.Amit Kumar	36 weeks	M	Asymptomatic	WNL
13	B.Amjad	33 weeks	M	Asymptomatic	GMH
14	B.Amra	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
15	B.Amravati	37 weeks	M	Asymptomatic	WNL
16	B.Anand	35 weeks	M	Hypotonia, Pallor, Refusal to take feeds, Convulsions	GMH
17	B.Aneeta	36 weeks	F	Hypotonia. Pallor. Tachycardia, Tachypnoea, Convulsions	GMH
18	B.Angoori	33 weeks	F	Hypotonia. Pallor. Tachycardia, Tachypnoea, Convulsions	GMH
19	B.Anita	34 weeks	M	Convulsions, Decerebrate Rigidity. Refusal to take feeds	PVL
20	B.Anju	35 weeks	M	Hypotonia. Pallor. Tachycardia, Refusal to take feeds. Convulsions	GMH
21	B.Ankit	37 weeks	M	Asymptomatic	WNL

22	B.Anuj	33 weeks	M	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
23	B.Anusuya	34 weeks	F	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
24	B.Archana	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
25	B.Arif	36 weeks	M	Asymptomatic	WNL
26	B.Arti	35 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
27	B.Asha	36 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
28	B.Asha	36 weeks	F	Asymptomatic	WNL
29	B.Asha Devi	37 weeks	F	Asymptomatic	WNL
30	B.Ashish	37 weeks	F	Asymptomatic	WNL
31	B.Ashok	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
32	B.Ashok	35 weeks	M	Asymptomatic	GMH
33	B.Atul	34 weeks	M	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
34	B.Aviral	36 weeks	M	Asymptomatic	WNL
35	B.Awastha Bai	37 weeks	F	Asymptomatic	WNL
36	B.Babita	34 weeks	F	Asymptomatic	GMH, Hydrocephalus
37	B.Babli	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
38	B.Babloo	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
39	B.Babu	34 weeks	M	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
40	B.Baldeo	35 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
41	B.Bano	35 weeks	M	Asymptomatic	GMH

42	B.Barai Lal	34 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
43	B.Basir	33 weeks	M	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
44	B.Beni Bai	37 weeks	M	Asymptomatic	WNL
45	B.Bhagwan Das	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
46	B.Bhagwanti	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
47	B.Bhagwati	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
48	B.Bhaiya Lal	37 weeks	F	Asymptomatic	WNL
49	B.Bharat Singh	37 weeks	M	Asymptomatic	WNL
50	B.Bharati	37 weeks	F	Asymptomatic	WNL
51	B.Bhawna	35 weeks	F	Asymptomatic	WNL
52	B.Bhoori	36 weeks	F	Hypotonia, Pallor, Refusal to take feeds, Convulsions	GMH
53	B.Bihari Lal	37 weeks	M	Asymptomatic	WNL
54	B.Bindu	36 weeks	M	Asymptomatic	WNL
55	B.Brij Bhan	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
56	B.Bulbul	33 weeks	F	Asymptomatic	GMH, Hydrocephalus
57	B.Chameli	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
58	B.Chanda	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
59	B.Chandana	36 weeks	F	Convulsions, Bulging Fontanelle	Hydrocephalus
60	B.Chandramukhi	35 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
61	B.Charan das	37 weeks	M	Asymptomatic	WNL
62	B.Chunni	33 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
63	B.Daisy	36 weeks	F	Asymptomatic	WNL

64	B.Damodar	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
65	B.Damodar	35 weeks	M	Asymptomatic	GMH, Hydrocephalus
66	B.Daulat	34 weeks	F	Hypotonia, Pallor, Tachycardia, Refusa to take feeds, Convulsions	GMH
67	B.Daya Khare	33 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
68	B.Dayaram	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
69	B.Deenanath	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
70	B.Deenu	35 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
71	B.Deepak	34 weeks	M	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
72	B.Devansh	37 weeks	M	Asymptomatic	WNL
73	B.Devendra Singh	35 weeks	M	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
74	B.Dhanku	37 weeks	M	Asymptomatic	WNL
75	B.Dheeraj	36 weeks	M	Asymptomatic	WNL
76	B.Dhirendra	34 weeks	M	Asymptomatic	GMH
77	B.Dino Bai	37 weeks	F	Asymptomatic	WNL
78	B.Draupadi	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
79	B.Fareed	36 weeks	M	Asymptomatic	WNL
80	B.Farha	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
81	B.Fatima	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH

82	B.Gariban	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
83	B.Gayatri	35 weeks	F	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
84	B.Geeta	34 weeks	M	Asymptomatic	GMH
85	B.Geeta	34 weeks	F	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
86	B.Ghasiram	34 weeks	M	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
87	B.Gomati	36 weeks	M	Asymptomatic	WNL
88	B.Gomti	35 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
89	B.Govind	33 weeks	M	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
90	B.Gulab Bai	37 weeks	M	Asymptomatic	WNL
91	B.Gulab Rao	37 weeks	M	Asymptomatic	WNL
92	B.Gulab Singh	37 weeks	M	Asymptomatic	WNL
93	B.Gurmez	36 weeks	M	Asymptomatic	WNL
94	B.Har Kumari	35 weeks	M	Hypotonia, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
95	B.Har Kunwar	35 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
96	B.Har Kunwari	35 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
97	B.Hardas	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
98	B.Hardas	36 weeks	M	Asymptomatic	WNL
99	B.Hari shanker	36 weeks	F	Asymptomatic	GMH
100	B.Harish	36 weeks	M	Asymptomatic	WNL
101	B.Harpal	36 weeks	M	Asymptomatic	WNL
102	B.Harsh Bahadur	36 weeks	M	Asymptomatic	WNL

103	B.Heera Bai	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
104	B.Himmat singh	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
105	B.Imran	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
106	B.Indira Devi	36 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
107	B.Indra	33 weeks	F	Asymptomatic	GMH
108	B.Ishu	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
109	B.Jagdish	37 weeks	M	Asymptomatic	WNL
110	B.Jagdish Singh	36 weeks	M	Asymptomatic	WNL
111	B.Jaidev	37 weeks	M	Asymptomatic	WNL
112	B.Jaidevi	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
113	B.Jaidevi	37 weeks	F	Asymptomatic	WNL
114	B.Jamvati	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
115	B.Janki	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
116	B.Janki Bai	32 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
117	B.Javed	35 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
118	B.Jitendra	34 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
119	B.Jyoti	37 weeks	M	Asymptomatic	WNL
120	B.Jyoti Singh	35 weeks	M	Asymptomatic	WNL
121	B.Kailashi	37 weeks	F	Asymptomatic	WNL
122	B.Kajal Rani	37 weeks	F	Asymptomatic	WNL
123	B.Kalicharan	34 weks	M	Hypotonia, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus

124	B.Kaloo	37 weeks	M	Asymptomatic	WNL
125	B.Kalpana	37 weeks	F	Asymptomatic	WNL
126	B.Kamla	35 weeks	F	Asymptomatic	GMH
127	B.Kamla Devi	32 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
128	B.Kamlesh	37 weeks	M	Asymptomatic	WNL
129	B.Kamleshi	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
130	B.Kanchan	37 weeks	F	Asymptomatic	WNL
131	B.Kapoor Chand	37 weeks	M	Asymptomatic	WNL
132	B.Kashi	35 weeks	M	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
133	B.Kashiram	33 weeks	M	Asymptomatic	GMH
134	B.Kastoori	37 weeks	F	Asymptomatic	WNL
135	B.Kastoori Bai	36 weeks	M	Asymptomatic	WNL
136	B.Kaushalya	36 weeks	F	Asymptomatic	WNL
137	B.Kavita	36 weeks	M	Asymptomatic	GMH, Hydrocephalus
138	B.Kesari	36 weeks	F	Asymptomatic	WNL
139	B.Khalida	35 weeks	F	Asymptomatic	GMH
140	B.Khelan	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
141	B.Kiran	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
142	B.Kiran	36 weeks	F	Asymptomatic	WNL
143	B.Kiran Sahu	37 weeks	F	Asymptomatic	WNL
144	B.Kishori	35 weeks	M	Asymptomatic	GMH
145	B.Kranti	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
146	B.Kunj Bano	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
147	B.Kusum Bai	37 weeks	F	Asymptomatic	WNL
148	B.Lad kunwar	37 weeks	F	Asymptomatic	WNL

149	B.Lakhan Singh	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
150	B.Lala Ram	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
151	B.Lallan	35weeks	M	Asymptomatic	GMH, Hydrocephalus
152	B.Laxmi	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
153	B.Laxmi	33 weeks	F	Asymptomatic	GMH
154	B.Laxmi Bai	36 weeks	F	Asymptomatic	WNL
155	B.Laxmi Narayan	36 weeks	M	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
156	B.Lekha	35 weeks	M	Asymptomatic	GMH, Hydrocephalus
157	B.Lokendra	35 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
158	B.Madeena	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
159	B.Madhavi	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
160	B.Malti	34 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
161	B.Malti	34 weeks	M	Asymptomatic	GMH, Hydrocephalus
162	B.Mamta	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
163	B.Mamta	34 weeks	F	Hypotonia, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
164	B.Mamta	35 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
165	B.Mamta Patel	36 weeks	M	Asymptomatic	WNL

167	B.Mangal Singh	34 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
168	B.Manish	36 weeks	M	Asymptomatic	WNL
169	B.Manjeet	35 weeks	F	Convulsions, Bulging Fontanelle	Hydrocephalus
170	B.Manorama	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
171	B.Mansha Ram	35 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
172	B.Mathews	37 weeks	F	Asymptomatic	WNL
173	B.Mathura prasad	34 weeks	M	Hypotonia, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
174	B.Maya Devi	36 weeks	F	Asymptomatic	WNL
175	B.Meena	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
176	B.Meena	35 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
177	B.Meenu	36 weeks	M	Asymptomatic	WNL
178	B.Meera	37 weeks	M	Asymptomatic	WNL
179	B.Mehmona	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
180	B.Minni	36 weeks	F	Asymptomatic	WNL
181	B.Mithila	35 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
182	B.Mithla	36 weeks	F	Asymptomatic	WNL
183	B.Mithun	37 weeks	M	Asymptomatic	WNL
184	B.Mohanlal	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
185	B.Montu	34 weeks	M	Asymptomatic	GMH, Hydrocephalus
186	B.Mukesh	35 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
187	B.Mukesh	35 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
188	B.Munna Khan	36 weeks	M	Asymptomatic	WNL
189	B.Munna Lal	36 weeks	M	Asymptomatic	WNL
190	B.Munni Devi	34 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL

191	B.Muskan	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
192	B.Najma	37 weeks	M	Asymptomatic	WNL
193	B.Najma Jahan	36 weeks	M	Asymptomatic	WNL
194	B.Nanadi	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea,, Bulging Fontanelle	GMH, Hydrocephalus
195	B.Nandini	37 weeks	F	Asymptomatic	WNL
196	B.Nandini Singh	37 weeks	F	Asymptomatic	WNL
197	B.Narmada	36 weeks	F	Asymptomatic	WNL
198	B.Naseema	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
199	B.Nasreen	35 weeks	M	Asymptomatic	GMH, Hydrocephalus
200	B.Nasreen	35 weeks	F	Convulsions Decerebrate Rigidity, Refusal to take feeds	PVL
201	B.Neelam	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
202	B.Neelam	36 weeks	F	Asymptomatic	WNL
203	B.Neena	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
204	B.Neeraj	35 weeks	M	Asymptomatic	GMH
205	B.Neeraj Devi	37 weeks	M	Asymptomatic	WNL
206	B.Nisha	35 weeks	F	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
207	B.Nitin	32 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
208	B.Noorjahan	36 weeks	F	Asymptomatic	WNL
209	B.Om Prakash	36 weeks	M	Asymptomatic	WNL
210	B.Panna Lal	37 weeks	M	Asymptomatic	WNL
211	B.Pappu	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
212	B.Parveen	36 weeks	M	Asymptomatic	WNL

213	B.Parwat Singh	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
214	B.Parwati	34 weeks	F	Asymptomatic	GMH, Hydrocephalus
215	B.Phool Singh	35 weeks	M	Asymptomatic	WNL
216	B.Phoolwati	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
217	B.Pinky	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
218	B.Pista	36 weeks	F	Asymptomatic	GMH
219	B.Poonam	35 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
220	B.Poonam	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
221	B.Prabha	37 weeks	M	Asymptomatic	WNL
222	B.Prabhu Dayal	36 weeks	M	Asymptomatic	WNL
223	B.Pramod Singh	36 weeks	M	Asymptomatic	WNL
224	B.Pratap	36 weeks	M	Asymptomatic	WNL
225	B.Pratima	37 weeks	F	Asymptomatic	WNL
226	B.Preeti	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
227	B.Preeti	36 weeks	M	Asymptomatic	WNL
228	B.Prem Bai	36 weeks	F	Asymptomatic	WNL
229	B.Prema	34 weeks	F	Asymptomatic	GMH, Hydrocephalus
230	B.Priyanka	37 weeks	F	Asymptomatic	WNL
231	B.Pushpa	35 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
232	B.Pushpa	35 weeks	F	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
233	B.Pushpa	36 weeks	M	Asymptomatic	WNL
234	B.Pushpa	37 weeks	F	Asymptomatic	WNL
235	B.Rabbo	32 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus

236	B.Radha	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
237	B.Radha	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
238	B.Radha	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH,
239	B.Radha	33 weeks	F	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
240	B.Radha	35 weeks	F	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
241	B.Radhe lal	36 weeks	M	Asymptomatic	WNL
242	B.Radhey Shyam	37 weeks	M	Asymptomatic	WNL
243	B.Raghunath	36 weeks	M	Asymptomatic	WNL
244	B.Raghuveer	36 weeks	M	Asymptomatic	WNL
245	B.Ragini	35 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
246	B.Rahisa	35 weeks	F	Asymptomatic	WNL
247	B.Rahul	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
248	B.Raj Dulari	34 weeks	F	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
249	B.Raj Kumar	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
250	B.Raj Kumar	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
251	B.Raj rani	36 weeks	M	Asymptomatic	WNL
252	B.Raja Bai	36 weeks	M	Asymptomatic	WNL
253	B.Raja Ram	34 weeks	M	Asymptomatic	GMH, Hydrocephalus

254	B.Rajesh	33 weeks	M	Asymptomatic	GMH, Hydrocephalus
255	B.Rajesh Kumar	37 weeks	M	Asymptomatic	WNL
256	B.Rajni	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
257	B.Rajni	35 weeks	F	Hypotonia, Bulging Fontanelle. Convulsions	GMH, Hydrocephalus
258	B.Raju	37 weeks	M	Asymptomatic	WNL
259	B.Rakhi	36 weeks	F	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
260	B.Raksha	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
261	B.Raksha	36 weeks	F	Convulsions, Bulging Fontanelle	Hydrocephalus
262	B.Ram Charan	34 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
263	B.Ram Devi	34 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
264	B.Ram Devi	36 weeks	F	Asymptomatic	WNL
265	B.Ram dhakeli	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
266	B.Ram Janki	34 weeks	M	Hypotonia, Pallor. Tachycardia, Tachypnoea, Convulsions	GMH
267	B.Ram Jeevan	36 weeks	M	Asymptomatic	WNL
268	B.Ram Khilawan	33 weeks	M	Hypotonia, Pallor. Tachycardia, Tachypnoea, Convulsions	GMH
269	B.Ram Kishan	37 weeks	M	Asymptomatic	WNL
270	B.Ram Kishore	34 weeks	M	Convulsions Decerebrate Rigidity. Refusal to take feeds	PVL
271	B.Ram Kumari	36 weeks	M	Hypotonia, Pallor. Tachycardia, Tachypnoea	GMH

272	B.Ram Kumari	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
273	B.Ram Prasad	36 weeks	F	Hypotonia. Pallor. Tachycardia. Tachypnoea, Convulsions	GMH
274	B.Ram Pyari	35 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds. Convulsions	GMH
275	B.Ram Rati	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
276	B.Ram Swaroop	35 weeks	M	Asymptomatic	WNL
277	B.Rama Mishra	37 weeks	F	Asymptomatic	WNL
278	B.Ramdevi	34 weeks	M	Asymptomatic	GMH
279	B.Ramshri	34 weeks	M	Asymptomatic	GMH, Hydrocephalus
280	B.Rani	36 weeks	F	Asymptomatic	WNL
281	B.Ranjana	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
282	B.Ranjana	37 weeks	F	Asymptomatic	WNL
283	B.Rashmi	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
284	B.Rati Ram	36 weeks	M	Asymptomatic	WNL
285	B.Ravi	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
286	B.Ravindra	35 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
287	B.Razia	37 weeks	F	Asymptomatic	WNL
288	B.Reena	34 weeks	M	Convulsions, Decerebrate Rigidity. Refusal to take feeds	PVL
289	B.Rekha	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
290	B.Renu Tripathi	36 weeks	M	Asymptomatic	WNL
291	B.Reshma	35 weeks	F	Asymptomatic	WNL
292	B.Richa	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
293	B.Rinki	36 weeks	F	Asymptomatic	WNL
294	B.Rinku	37 weeks	M	Asymptomatic	WNL

295	B.Rohit	37 weeks	M	Asymptomatic	WNL
296	B.Roshan	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
297	B.Roshni	37 weeks	F	Asymptomatic	WNL
298	B.Sadhna	35 weeks	M	Asymptomatic	GMH
299	B.Safia	35 weeks	M	Asymptomatic	GMH
300	B.Sahida	37 weeks	F	Asymptomatic	WNL
301	B.Sahil	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
302	B.Sahil	35 weeks	M	Asymptomatic	WNL
303	B.Saira	34 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH, Hydrocephalus
304	B.Sajjan	37 weeks	M	Asymptomatic	WNL
305	B.Samini	35 weeks	F	Convulsions, Bulging Fontanelle	Hydrocephalus
306	B.Sampat	35 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
307	B.Sandhya	36 weeks	F	Asymptomatic	WNL
308	B.Sangam	37 weeks	M	Asymptomatic	WNL
309	B.Sangeeta	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
310	B.Sanjana	34 weeks	F	Asymptomatic	GMH
311	B.Sanjay	34 weeks	M	Asymptomatic	GMH, Hydrocephalus
312	B.Sanju	35 weeks	M	Decerebrate Rigidity. Refusal to take feeds	PVL
313	B.Santosh Kumari	37 weeks	F	Asymptomatic	WNL
314	B.Santoshi	34 weeks	F	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
315	B.Sapna	34 weeks	F	Hypotonia, Bulging Fontanelle, Convulsions	GMH. Hydrocephalus
316	B.Saroj	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
317	B.Saroj	33 weeks	F	Asymptomatic	GMH
318	B.Sarojini	36 weeks	F	Asymptomatic	WNL
319	B.Saurabh	35 weeks	M	Convulsions Decerebrate Rigidity. Refusal to take feeds	PVL

320	B.Savitri	33 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
321	B.Savitri	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
322	B.Seema	36 weeks	M	Asymptomatic	WNL
323	B.Shahabat	34 weeks	M	Asymptomatic	GMH
324	B.Shahanshah	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
325	B.Shahin	36 weeks	M	Asymptomatic	GMH, Hydrocephalus
326	B.Shahjahan	37 weeks	M	Asymptomatic	WNL
327	B.Shahnaz	37 weeks	M	Asymptomatic	WNL
328	B.Shailendra	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
329	B.Shakuntala	35 weeks	F	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
330	B.Shakuntala	37 weeks	F	Asymptomatic	WNL
331	B.Shaleen	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
332	B.Shaligram	33 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
333	B.Shalini	34 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
334	B.Shanno	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
335	B.Shanti	37 weeks	F	Asymptomatic	WNL
336	B.Sharda	35 weeks	M	Hypotonia, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
337	B.Sharda	37 weeks	F	Asymptomatic	WNL
338	B.Shashi	34 weeks	F	Decerebrate Rigidity, Refusal to take feeds	PVL
339	B.Sheela	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea,	GMH

				Convulsions	
340	B.Sheela	35 weeks	F	Asymptomatic	GMH
341	B.Sheelu	35 weeks	F	Asymptomatic	WNL
342	B.Sheikh	35 weeks	M	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
343	B.Shiv Charan	36 weeks	M	Asymptomatic	WNL
344	B.Shiv Kumari	36 weeks	F	Asymptomatic	WNL
345	B.Shivani	36 weeks	M	Asymptomatic	WNL
346	B.Shivnath	36 weeks	M	Asymptomatic	WNL
347	B.Shubha	35 weeks	F	Asymptomatic	WNL
348	B.Shyam Bai	36 weeks	F	Asymptomatic	WNL
349	B.Shyam Kureshi	37 weeks	M	Asymptomatic	WNL
350	B.Shyam singh	34 weeks	M	Asymptomatic	GMH
351	B.Siya Dulari	35 weeks	F	Hypotonia, Bulging Fontanelle, Convulsions	GMH, Hydrocephalus
352	B.Siya Rani	33 weeks	M	Hypotonia, Pallor, Bulging Fontanelle, Refusal to take feeds, Convulsions	GMH, Hydrocephalus
353	B.Snehlata	35 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH
354	B.Sona	35 weeks	F	Asymptomatic	WNL
355	B.Sri Niwas	34 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
356	B.Sri Ram	35 weeks	M	Asymptomatic	WNL
357	B.Subhash	37 weeks	M	Asymptomatic	WNL
358	B.Sukhlal	36 weeks	M	Convulsions, Bulging Fontanelle	Hydrocephalus
359	B.Suman	33 weeks	M	Asymptomatic	GMH
360	B.Sumitra	32 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Convulsions	GMH, Hydrocephalus
361	B.Suneeta	34 weeks	F	Asymptomatic	GMH, Hydrocephalus
362	B.Sunita	36 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
363	B.Sunita	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
364	B.Sushan	34 weeks	M	Asymptomatic	GMH,

					Hydrocephalus
365	B.Susheela	37 weeks	M	Asymptomatic	WNL
366	B.Sushma	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
367	B.Sushma	33 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
368	B.Tulsidas	35 weeks	M	Asymptomatic	GMH
369	B.Uma	36 weeks	F	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH
370	B.Uma	36 weeks	F	Asymptomatic	WNL
371	B.Uma Sharma	36 weeks	F	Asymptomatic	WNL
372	B.Umesh	35 weeks	M	Decerebrate Rigidity, Refusal to take feeds	PVL
373	B.Umesh Kumar	34 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea	GMH, Hydrocephalus
374	B.Vandana	33 weeks	M	Hypotonia, Pallor, Tachycardia, Tachypnoea, Bulging Fontanelle	GMH, Hydrocephalus
375	B.Veer Singh	36 weeks	F	Asymptomatic	WNL
376	B.Vijay	36 weeks	M	Asymptomatic	WNL
377	B.Vimla Singh	37 weeks	F	Asymptomatic	WNL
378	B.Vineeta	36 weeks	M	Hypotonia, Pallor, Tachycardia, Refusal to take feeds, Convulsions	GMH
379	B.Vineeta	36 weeks	M	Asymptomatic	WNL
380	B.Vinita	35 weeks	F	Asymptomatic	GMH, Hydrocephalus
381	B.Vinodini	35 weeks	F	Asymptomatic	GMH
382	B.Waris	34 weeks	M	Hypotonia, Pallor, Bulging Fontanelle. Refusal to take feeds, Convulsions	GMH, Hydrocephalus
383	B.Zahida	34 weeks	F	Convulsions, Decerebrate Rigidity, Refusal to take feeds	PVL
383	B.Zubeida	34 weeks	F	Decerebrate Rigidity. Refusal to take feeds	PVL
384	B.Zuber	37 weeks	M	Asymptomatic	WNL

S.No	Name	Age	Sex	Symptoms	Sonographic Finding
1	Aarti	11 months	F	Fever. Convulsions. Refusal to take feeds, Neurological Deficit	Echogenic Sulci. Hydrocephalus
2	Abdul Saeed	11 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
3	Abhimanyu	6 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
4	Abhishek	29 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
5	Adil	2 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydranencephaly
6	Ahilya	2 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
7	Ajab	4 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
8	Ajay	9 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
9	Akila	11 days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus
10	Amit	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
11	Amlesh	11 months	M	Fever, Refusal to take feeds, , Hypotonia	Echogenic Sulci,
12	Amra	2 months	F	Fever, Convulsions	WNL
13	Aneeta	23 days	F	Convulsions, Pallor	WNL
14	Anita	12 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
15	Anita	6 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci Cerebritis
16	Anju	28 days	F	Bulging Fontanelle, Hypotonia, Convulsions, Neurological Deficit	Cystic Encephalomalacia
17	Anju	9 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
18	Archana	10 days	M	Convulsions, Pallor	GMH, Hydrocephalus

19	Arjun	4 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
20	Arvind	10 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
21	Arvind	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
22	Ashish	21 days	M	Bulging Fontanelle, Hypotonia, Convulsions Neurological Deficit	Porencephaly
23	Ashish	6 months	M	Convulsions	WNL
24	Ashok	14cdays	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
25	Ashok Kumar	24 days	M	Bulging Fontanelle, Hypotonia, Convulsions Neurological Deficit,	Porencephaly
26	Babita	4 months	F	Convulsions	WNL
27	Babloo	3 days	M	Convulsions, Pallor	GMH, Hydrocephalus
28	Babu	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
29	Balram	4 months	M	Convulsions	WNL
30	Bantu	11 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
31	Bhaggo	11 months	F	Fever, Convulsions, Neurological Deficit	Cerebral Abscess
32	Bhagwan Das	4 days	M	Convulsions, Pallor	GMH
33	Bhagwati	4 days	M	Bulging Fontanelle, Hypotonia, Convulsions Neurological Deficit	Porencephaly
34	Bharti	9 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
35	Bhoora	5 months	M	Fever, Convulsions, Neurological Deficit	Cerebral Abscess
36	Bhupendra	6 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
37	Bhupendra	9 months	M	Fever, Convulsions, Neurological Deficit	Cerebral Abscess

38	Bindwasa	7 days	M	Neurological Deficit, Pallor	Hydrocephalus
39	Chand Bano	4 days	F	Bulging Fontanelle, Hypotonia. Neurological Deficit	Hydrocephalus
40	Chanda	7 days	M	Convulsions, Pallor	GMH, Hydrocephalus
41	Chandan	5 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
42	Chaturbhuj	11 days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus
43	Chhakki	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
44	Chhanga	11 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
45	Chhote lal	10 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
46	Chhotu	5 months	M	Convulsions	WNL
47	Chintu	9 months	M	Convulsions	WNL
48	Deepa	10 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci Cerebritis
49	Deepak	7 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
50	Deependra	11 months	M	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci Cerebritis
51	Devmani	25 days	F	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
52	Dharmendra	2 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
53	Diljit	28 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
54	Dinesh	9 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
55	Draupadi	7 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
56	Draupadi	3 days	F	Convulsions, Pallor	Hydrocephalus
57	Durji	3 days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus

58	Gabbar	4 months	M	Convulsions	WNL
59	Ganpat	22 days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus
60	Gautam	10 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Cystic Encephalomalacia
61	Gaya Prasad	11 months	M	Fever, Convulsions Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
62	Geeta	13 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
63	Giri raj	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
64	Girija	5 months	F	Fever, Convulsions	WNL
65	Gopal	3 months	M	Convulsions	WNL
66	Gopi	6 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
67	Gore lal	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
68	Guddi	11 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
69	Guddu	5 months	M	Convulsions	WNL
70	Habeeb	13 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
71	Hasmukhi	25 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
72	Hemant	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
73	Hridesh	25 days	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Cystic Encephalomalacia
74	Imam	8 months	M	Fever, Convulsions, Neurological Deficit	Cerebral Abscess
75	Imtiaz	3 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
76	Indira	8 months	F	Fever, Convulsions	WNL
77	Jai Kunwar	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus

78	Jaidevi	4 days	M	Convulsions, Pallor	GMH, Hydrocephalus
79	Jyoti	7 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
80	Kalka Prasad	5 months	M	Convulsions	WNL
81	Kallu	11 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
82	Kalpana	4 months	F	Convulsions	WNL
83	Kamini	11.5 mths	F	Convulsions, Neurological Deficit	Mass Lesion Hydrocephalus
84	Kamlesh	18 days	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
85	Kanak	9 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
86	Kanchan	25 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
87	Kapoori	2 months	F	Fever, Convulsions	WNL
88	Karan	8 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
89	Karishma	10 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
90	Kaushalya	7 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
91	Keshkali	5 months	F	Convulsions	WNL
92	Khera	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
93	Khilai	28 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
94	Khilan	5 months	F	Fever, Convulsions	WNL
95	Kiran	3 days	F	Convulsions, Pallor	GMH, Hydrocephalus
96	Kishan	11 months	M	Fever. Refusal to take feeds, Neurological Deficit , Hypotonia	Hydrocephalus
97	Kishori	11 months	M	Fever, Convulsions	WNL
98	Komal Singh	11 months	M	Convulsions	WNL
99	Kranti	3 days	M	Convulsions, Pallor	GMH
100	Krishna	17 days	F	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
101	Kunti	13 days	F	Convulsions, Pallor	WNL
102	Lakshmi	1 day	M	Convulsions, Pallor	GMH

103	Lala Ram	21 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
104	Lalita	4 months	F	Convulsions	WNL
105	Leela	5 days	M	Neurological Deficit, Pallor	Hydrocephalus
106	Maan Singh	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
107	Madeena	6 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
108	Madhavi	2 days	M	Convulsions, Pallor	GMH
109	Mahesh	5 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Cystic Encephalomalacia
110	Mallu	5 months	M	Convulsions	WNL
111	Malti	24 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
112	Malti	1 month	F	Fever, Convulsions	WNL
	Malvika	9 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
113	Mamta	4 days	M	Convulsions, Pallor	GMH, Hydrocephalus
114	Mandavi	15days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus
115	Manish	7 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
116	Manisha	9 months	F	Convulsions, Neurological Deficit	Mass Lesion
117	Manju	18 days	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
118	Manku	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
119	Mannu	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
120	Manoj	12 months	M	Convulsions, Neurological Deficit	Mass Lesion Hydrocephalus
121	Manorama	2 months	F	Fever, Convulsions	WNL
122	Matadin	25 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
123	Maya	8 months	F	Fever, Convulsions	WNL

124	Mayank	6 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
125	Meena	2 months	F	Fever, Convulsions	WNL
126	Meera	6 months	F	Fever, Convulsions	WNL
127	Mehraj	10 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
128	Mini	7 months	F	Fever, Convulsions	WNL
129	Mithila	8 months	F	Fever, Convulsions	WNL
130	Munna Raja	6 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
131	Muskan	5 months	F	Fever, Hypotonia	Echogenic Sulci Cerebritis
132	Nancy	10 months	F	Fever, Convulsions, Neurological Deficit	Cerebral Abscess
133	Nandini	5 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
134	Narayan	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
135	Naseema	10 months	F	Fever, Convulsions, Neurological Deficit	Cerebral Abscess
136	Natthu	6 months	M	Convulsions	WNL
137	Nazba	7 months	F	Fever, Convulsions	WNL
138	Nazma	2 months	F	Fever, Convulsions	WNL
139	Nazneen	8 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci Cerebritis
140	Neelam	14 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
141	Neelam	27 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
142	Neelam	7 months	F	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
143	Neelam	7 months	F	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
144	Neena	7 days	M	Convulsions, Pallor	GMH, Hydrocephalus
145	Neetu	9 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci Cerebritis
146	Nitin	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus

147	Noorjahan	11 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
148	Om Prakash	5 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
149	Pankaj	11 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
150	Parwati	25 days	F	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
151	Pinky	18 days	F	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
152	Poonam	5 months	F	Fever, Convulsions	WNL
153	Pratibha	9 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci
154	Preeti	4 months	F	Convulsions, Neurological Deficit	Mass Lesion Hydrocephalus
155	Preeti	11 months	F	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
156	Prema	7 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
157	Priyank	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
158	Prothi	8 months	F	Fever, Convulsions, Refusal to take feeds. Neurological Deficit. Hypotonia	Echogenic Sulci, Hydrocephalus
159	Pushpa	3 months	F	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
160	Pushpendra	9 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
161	Radhey Lal	7 months	M	Convulsions	WNL
162	Rafique	8 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
163	Raghuveer Giri	22 days	M	Bulging Fontanelle. Hypotonia, Neurological Deficit	Hydrocephalus

164	Raghuveer Singh	17 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
165	Raghuver	4 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
166	Rahul	2 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
167	Rahul	15 days	M	Bulging Fontanelle, Convulsions, Neurological Deficit	Mass Lesion
168	Rahvas	8 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
169	Raj Kumari	23 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
170	Raj Kumari	23 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
171	Raj kumari	18 days	M	Neurological Deficit, Pallor	Hydrocephalus
172	Raja	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
173	Rajeshwari	4 months	F	Fever, Convulsions	WNL
174	Rajneesh	4 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
175	Rajni	23 days	M	Bulging Fontanelle, Hypotonia	Hydrocephalus
176	Raju	4 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
177	Raksha	8 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
178	Ram Shran	4 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Cystic Encephalomalacia
179	Rama Devi	15 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
180	Ramdeen	4 months	M	Convulsions	WNL
181	Ramkali	6 months	F	Fever, Convulsions	WNL
182	Ramku	8 months	F	Convulsions	WNL
183	Rani	3 days		Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus

184	Rani Soni	12 days	F	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
185	Ranu	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
186	Rashmi	21 days	M	Convulsions, Pallor	WNL
187	Rashmi	2 days	M	Convulsions, Pallor, Neurological Deficit	Hydrocephalus
188	Rasoodan	6 days	F	Bulging Fontanelle, Hypotonia, Convulsions, Neurological Deficit	Cystic Encephalomalacia
189	Ravindra	4 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
190	Razia	10 monhs	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
191	Reena	17 days	M	Convulsions, Pallor	WNL
192	Rekha	3 months	F	Fever, Convulsions	WNL
193	Renu	11 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
194	Rishi	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
195	Rohit	5 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
196	Roshni	6 months	F	Convulsions	WNL
197	Rukmani	7 months	F	Convulsions	WNL
198	Sameer	24 days	M	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
199	Samiksha	9 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
200	Samina	10 months	F	Fever, Refusal to take feeds, Hypotonia	Echogenic Sulci
201	Sandeep	2 days	M	Bulging Fontanelle, Hypotonia, Convulsions, Neurological Deficit	Cystic Encephalomalacia
202	Sandhya	28 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus

227	Shilpi	3 months	F	Convulsions	WNL
228	Shivi	10 monhs	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
229	Shyamu	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
230	Simran	6 days	F	Bulging Fontanelle, Pallor, Neurological Deficit	Hydrocephalus
231	Siya Rani	9 months	F	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
232	Snehlata	7 days	F	Convulsions, Pallor	GMH
233	Sona bai	19 days	M	Convulsions, Pallor	WNL
234	Sonu	6 months	M	Convulsions	WNL
235	Sonu	8 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
236	Suman	3 days	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
237	Suman	3 months	F	Fever, Convulsions	WNL
238	Sumitra	5 months	F	Convulsions	WNL
239	Suneel	8 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
240	Sunita	18 days	F	Bulging Fontanelle, Hypotonia	Hydrocephalus
241	Sunita	2 days	F	Convulsions, Pallor	GMH
242	Surendra	3 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
243	Sushma	2 days	F	Convulsions, Pallor	GMH, Hydrocephalus
244	Swami	4 months	M	Fever, Refusal to take feeds, Neurological Deficit, Hypotonia	Hydrocephalus
245	Taranum	18 ays	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
246	Teenu	11 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus

247	Thakur Das	9 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit, Hypotonia	Echogenic Sulci, Hydrocephalus
248	Tiggu	7 months	M	Convulsions	WNL
249	Tulsi	5 days	F	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
250	Uma	4 months	F	Fever. Convulsions	WNL
251	Umesh Kumar	5 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydranencephaly
252	Urmila	7 months	F	Fever. Convulsions	WNL
253	Usha	6 months	F	Fever. Convulsions	WNL
254	Vandana	3 months	F	Fever, Convulsions	WNL
255	Vaseem	5 months	M	Convulsions	WNL
256	Vedant	5 months	M	Fever. Convulsions	WNL
257	Veenu	10 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
258	Vidya	11 months	F	Fever, Convulsions	WNL
259	Vijay Lakshmi	27 days	F	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
260	Vijendra	8 months	M	Convulsions	WNL
261	Vikrant	24 days	M	Bulging Fontanelle, Hypotonia, Pallor	Hydrocephalus
262	Vimala	4 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Porencephaly
263	Vinita	3 months	F	Fever, Convulsions	WNL
264	Vinod	9 months	M	Fever, Refusal to take feeds, Neurological Deficit. Hypotonia	Hydrocephalus
265	Vishal	9 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
266	Wasim	7 months	M	Fever, Convulsions, Refusal to take feeds, Neurological Deficit	Echogenic Sulci, Hydrocephalus
267	Yogesh	21 days	M	Bulging Fontanelle, Hypotonia, Neurological Deficit	Hydrocephalus
268	Yusuf	7 months	M	Fever, Hypotonia	Echogenic Sulci Cerebritis
269	Zarina	10 months	F	Fever, Hypotonia	Echogenic Sulci, Cerebritis